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16 ABSTRACT

This literature review covered 464 scientific papers with categories on Reviews and Symposia, Dissolved Gases, Salinity, pH, Insecticides, Herbicides, Piscicides, Inorganics, Organics, Domestic Pollutants, Radioactive Pollutants, and Other Pollutants. The review includes a tabulation of numerical results of toxicity testing.

17 KEY WORDS AND DOCUMENT ANALYSIS

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JOURNAL ARTICLE

Literature Review

Effects of pollution on freshwater fish

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REVIEWS AND SYMPOSIA

Fairchild *et al.*[1] summarized the literature from 1972 to 1975 on the effects of chlordane and heptachlor on freshwater fish and other aquatic life. Low levels of chlordane and heptachlor were shown to be toxic and residues of chlordane, heptachlor, and heptachlor epoxide were widespread. Grant[2] reviewed information on the toxicity of endrin to freshwater aquatic organisms and the distribution of endrin in freshwater systems. These and other investigations were the basis for a recommended maximum concentration of 0.002 µg endrin/l in water for the protection of aquatic organisms. The U. S. Department of the Interior Water Resources Scientific Information Center[3] published a bibliography on DDT in water which contained abstracts of 480 papers on accumulation and effects on freshwater fishes.

Ahmed[4] reviewed the continuing problem of polychlorinated biphenyl (PCB) contamination, particularly emphasizing the contamination of sport and commercial fish in the U. S., sediment accumulation, toxic effects of PCBs, and control of PCBs. The problem of PCBs in the environment was discussed by Josephson[5] with emphasis on the residue problem in the fisheries of the Great Lakes, medical effects, contamination of food, and proposed alternatives to PCBs. Kleinert[6] described the studies which confirmed the presence of PCBs in fish in Lake Michigan and other state waters, and in some detail discussed the results

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of the search for the sources of the PCBs. In another general review article, Jackson[7] described the problems of PCBs in the environment and residues in fish tissue.

Pesticide-induced changes in the liver of fishes were reviewed by Couch[8] and provided as a representative index to the published histological works on fishes experimentally exposed to pesticides. Most of these studies involved freshwater fishes, particularly the rainbow trout (*Salmo gairdneri*) and bluegill (*Lepomis macrochirus*), and demonstrated that liver damage occurs in both acutely lethal and chronic sublethal exposures of fish to certain pesticides. Jhingran's[9] review of the pollution in rivers, reservoirs, and estuaries in India included reports of complete loss of fish life, primarily from increased use of pesticides in agriculture. The Water Research Centre's Stevenage Laboratory, England[10] compiled a comprehensive list of polluting substances arranged in order of general toxicity and keyed as to source of pollutant, concentration of pollutant in the sample (when figures were available), and analytical techniques used. Included were polynuclear aromatic hydrocarbons, cyanides, phenols, ethers, organohalogens, and heterocyclics. Suffet[11] presented a review of the 1974 literature on water pollution which included toxicity and biological effects of detergents, petroleum contaminants, biocides, PCBs, herbicides, and chlorinated hydrocarbons on aquatic organisms.

The European Inland Fisheries Advisory Commission[12] reviewed the chemistry and toxicity of copper with regard to freshwater aquatic life. Lethal and sublethal effects in the laboratory and the field were found to vary greatly with a wide variety of physical and chemical characteristics of water. Approximate maximum annual 50 and 95 percentile concentrations of soluble Cu for the protection of rainbow trout, and probably other salmonid species, ranged from 1.0 to 118 μg Cu/l, dependent upon water hardness. These water quality criteria were derived using 0.05 and 0.3 of the threshold TL_m values for the 50 and 95 percentile concentrations, respectively. A section on marine and freshwater fishes was included in a review by Leland *et al.*[13] on heavy metals and related trace elements, noting that concentrations of trace elements occurring naturally in fish tissues were not well defined. Zitko[14] reviewed the toxicity and pollution potential of Tl and reported that Tl was slightly more acutely toxic to mammals than Hg, and as acutely toxic as Cu to fish, but little was known about the chronic toxicity and tissue levels resulting from chronic exposure. Dandurand[15] presented a comprehensive and critical review on the toxicity to fish of simple and complex cyanides and related compounds. Effects of sublethal levels of free cyanide on growth, food consumption, swimming ability, behavior, and avoidance reactions of fish to the toxicant were summarized. Interactions of free cyanide with other toxic water pollutants were also considered. Results of studies on the effects of fluoride on freshwater fish were included in a review on fluoride pollution by Groth.[16] Short-term lethal effects occurred at concentrations as low as 3 mg/l in rainbow trout, which was considered a sensitive species. Fish have the ability to accumulate elevated fluoride levels in their skeletons, thus introducing the contaminant into the diet of fish-eating predators.

Saalfeld and Howell[17] reviewed the progress of sea lamprey (*Petromyzon marinus*) control in the Great Lakes using 3-trifluoromethyl-4-nitrophenol (TFM), a selective chemical for lampreys. Federal fishery agencies of the U. S. and Canada began chemical treatment in streams tributary to Lake Superior in 1958 and have extended the treatment program as funds become available. The degree of control in Lake Superior and Lake Michigan has made it possible to begin rehabilitation of valuable fish stocks. Menzie and Hunn[18] reviewed the testing of TFM prior to its use as a lampricide in the Great Lakes, and reported that it was not subject to biomagnification, and that tissue levels in fish declined rapidly and approached the detection limits (0.01 mg/l) by the end of 4 days. Residues in Lake Superior fish were not detected. Marking[19] presented a protocol for determining and evaluating the toxicity of chemicals to aquatic organisms since efforts are being made to standardize registration procedures for compounds used as piscicides in fisheries. A discussion was presented that identified different types of toxicity tests, suggested preferred species, and identified and assessed factors that influence the toxicity of chemicals to fish. Cumming[20] tabulated ten major piscicides that have been used extensively in fish population rehabilitations. Only four piscicides are presently registered: antimycin and rotenone as general fish toxicants, and TFM and Bayluscide as selective lampricides. Burress[21] compared the effectiveness of rotenone and antimycin for selective thinning operations in black bass (*Micropterus* sp.) management, and reported that rotenone was the toxicant of choice where catfish (*Ictalurus* sp.) must be removed, or in waters having a

high pH. Antimycin was less toxic to bass than to most commonly associated forage fish. Hurlbut [3] noted that pesticide-induced population increases occurred from predator removal or increased food supply, and removal of fish by fish toxicants has often been followed by increases in invertebrate populations. Times and places were documented where this occurred after the use of rotenone and toxaphene in aquatic ecosystems.

A bibliography containing 191 references was compiled by Mattice *et al.*[4] on the chemistry and effects of chlorine in aquatic systems, both freshwater and marine. Chlorine residuals were recorded, as well as methods of chlorine determination, and physical conditions under which the experiments were run. Time-dependent acute and chronic threshold concentrations were presented for freshwater and marine environments. Brungs [5] compiled a continuation of an earlier review on the effects of wastewater and cooling water chlorination on aquatic life. Several topics were discussed, one being aquatic life criteria with the recommendation from EPA that the exclusive use of Cl_2 for disinfection should not be continued where protection of aquatic life was of primary consideration. Additional topics discussed the acute and chronic effects of short-term intermittent and continuous exposure of freshwater fish to total residual chlorine. Rosenthal [6] compiled a bibliography on ozone and found that only a few papers dealt with biological effects of ozone on aquatic organisms. Ozonation has become a most beneficial standard method for water treatment in home aquaria, and fisheries biologists are increasingly considering the application of ozone in water treatment and re-use systems.

The bioaccumulation factors for tritium and isotopes of Sr, Cs, I, Mn, and Co were collected from over 200 selected papers and analyzed and discussed by Vanderploeg *et al.*[7] Results of field studies on freshwater organisms were emphasized, including fish, to develop simple models. Patzer [8] reviewed the literature on the biological concentration of radionuclides in freshwater and marine fish and invertebrates. Concentration factors were tabulated and various radiation dose models were developed to determine the potential impact on humans. Volchok [9] acknowledged in a review that there has been little research on the carcinogenic potential of radioactive materials on freshwater fish, but summarized the available information on the aquatic distribution of those materials and the occurrence and concentration of significant radionuclides in aquatic animals.

Chemical carcinogens in aquatic environments were identified in a review by Kraybill.[10] These chemical contaminants have produced toxic effects in fish, causing certain diseases and pathological lesions, including the development of tumors. Speculation was that extensive investigation of the aquatic environment may play an important role in the epidemiology of cancer. Vertebral fractures in fish were reviewed by Bengtsson [11] in relation to water pollution. Chlorinated hydrocarbons, organophosphorus pesticides, and heavy metals have experimentally caused vertebral damage. Such damage in wild fish populations could be an early warning of water pollution.

Machlis [12, 13] prepared four literature reviews and bibliographies on the effects of hydroelectric development on the biology of lake whitefish (*Coregonus clupeaformis*), northern pike (*Esox lucius*), yellow walleye (*Stizostedion vitreum vitreum*), and lake trout (*Salvelinus namaycush*), including effects of fluctuating water levels and impoundment. Cairns *et al.*[14, 15] reviewed the literature on temperature effects on toxicity of chemicals to aquatic organisms and found field data on this relationship to be almost non-existent. If water quality management programs are to be developed for steam-electric power plants, more information is needed, since some heated waste contains chlorine and heated water may be discharged in the vicinity of toxic chemicals, in which cases the toxic response may be increased at the higher temperature. The U. S. Energy Research and Development Administration [16] prepared a bibliography on environmental studies at the Savannah River Plant, S. C., listing research papers pertinent to that region. The 702 citations were arranged by category and included a section on pollution effects on freshwater fish. McKim *et al.*[17] presented an extensive review of the 1975 literature on the effects of pollution on freshwater fish. Metals, pesticides, and domestic and industrial wastes were included, as well as the effects of water quality (salinity, pH, and O_2). Davis [18] summarized the literature on the responses of salmonids to sublethal threshold concentrations of neutralized whole bleached kraft mill effluent, kraft bleaching, and the production of unbleached pulp. Sublethal responses included effects on growth, behavior, and temperature tolerance, and large differences in sensitivity were not evident between salmonid species. The toxicant-wildlife complex as related to aquatic species was reviewed

by Cope.[7] This major portion of vertebrate wildlife will be increasingly exposed to a wide variety of man-made and natural chemicals. Results of the review suggested that bioconcentration of stable substances usually dependent upon the fat content of the individuals, may not be strongly affected by metabolism, will not inevitably lead to biomagnification, and will become increasingly predictive. Basic population processes for fishes were reviewed by McFadden,[8] and the ways in which environmental impacts were reflected in changes in population parameters was explored. The paper concluded with a demonstration of the use of fish population data in decision-making, where the question of acceptability of environmental impacts was dealt with on at least a partially ecological basis. Kleinkopf[9] discussed the need to determine the sublethal responses of fish to stress, including pollution, and recommended the study of behavioral responses, such as locomotor behavior and orientation. A general discussion was presented of the physical testing systems that have been used to evaluate Cu, Cu with temperature, parathion, food odor, and rates of water flow. Sprague[10] tabulated the current status of sublethal tests of pollutants on aquatic organisms and concluded that a good experimental basis for water quality criteria has not been achieved as yet. The most sensitive and productive experiments seemed to be chronic tests on reproductive cycles and early stages of organisms.

The International Joint Commission (IJC)[11] published the proceedings of a workshop on the correlation of chemical structure-activity with toxicity and bioconcentration in relation to aquatic organisms. Several papers discussed this correlation with data on fish and surfactants, DDT analogs, numerous other pesticides, and organic chemicals. The IJC[12] sponsored another workshop on the toxicity of various metal forms in natural water to freshwater organisms. Metal speciation in relation to the toxicity of several metals, including Cu, Zn, and Hg to fish was discussed by several authors. Proceedings of a conference held at Oak Ridge National Laboratory[13] set down the present state of knowledge concerning aqueous chlorination, with particular emphasis on chlorinated organic compounds and associated biomedical and environmental effects, including those on freshwater organisms. The U. S. Environmental Protection Agency Office of Toxic Substances[14] published the proceedings of the conference on "Environmental Aspects of Chemical Use in Well-Drilling Operations." Papers were included on the acute toxicity of weighting materials (barite, calcite, and siderite) in drilling fluids and drilling mud deflocculants (phosphates, tannins, ground lignites, and lignosulfonates) to mollies (*Poecilia latipinna*) in fresh water. In addition, the acute toxicity of muds taken at various well depths was determined using rainbow trout, and the toxicity of organic components of drilling muds to juvenile Atlantic salmon (*Salmo salar*) was discussed. The proceedings of a conference held in 1973 were edited by Brenkel[15] and included a section on toxicology and physiological response to heavy metals by fish.

WATER QUALITY

Dissolved gases. Seasonal mortalities of stocked underyearling rainbow trout (*Salmo gairdneri*) were studied by Ayles *et al.*[16] in eight small eutrophic Canadian prairie lakes. Two periods of mortality were observed. One, shortly after stocking, was believed largely to seasonal algal blooms. Of the several parameters measured (DO, ammonia, temperature, algal biomass, chlorophyll *a*, and pH), DO was designated the most likely single causative agent. Synergism with temperature and un-ionized ammonia was also believed involved. Summer-to-summer (9-month) predictions for summer fish kills suggested that fish should not be stocked in lakes with chlorophyll *a* concentrations above 100 μg/l, and DO should be from 6 to 15 mg/l (Barica[17]). Secchi disc transparency should exceed 0.4 m for *Aphanizomenon flosaquae* blooms and 0.5 for other blooms. Winter-to-summer (3-month) predictions suggested that during February to early March NH_3-N concentrations should not exceed 1 000 μg/l, and DO during this period should not exceed 1 mg/l. Complete aerate winter conditions were considered desirable in summer trout production ponds.

Vertical movements of rainbow trout through DO-deficient waters were revealed by Berns.[18] Marked trout from a group pumped into the hypolimnion of a stratified 12-m lake were later netted at the surface, in spite of a 5-m DO-deficient intermediate zone with only 0.5 to 0.7 mg DO/l. Members of another group released at the surface were also caught in nets set on the bottom. Casselman and Harvey[19] studied naturally-occurring winter kills caused by low DO on northern pike (*Esox lucius*) in two Canadian lakes. A minimum DO of 1.1 mg/l was observed in Smoky Hollow Lake and 0.8 mg/l in Wickett Lake. Differential mor-

tabilities were reported by age, growth rate, and sex of the northern pike sampled, with fish more than 6 years old and gametic ripening females being less tolerant than younger fish. Intensive pellet feeding in carp (*Cyprinus carpio*) rearing ponds was reported by Bachtheluss [12] to have reduced the importance of pond area as a food supplier and increased the importance of available O_2 because of overcrowding. Havelka [13] found that cascades from dams on the Vltava River in the Prague area of Czechoslovakia improved the O_2 content in the water to such an extent that trout fisheries were established where only coarse fish were present before the dams were installed.

Critical O_2 concentrations and O_2 consumption rates of seven species of freshwater fishes were studied by Khyatkurin and Yarzhombek.[14] Consumption rates were similar within a species over size ranges as great as 0.05 to 30 g. Critical O_2 concentrations were found to increase among the species studied in the following order: carp, white amur, grass carp, coho salmon, rainbow trout, stellate sturgeon, and lena sturgeon. Galkina [15] reported 1.05 ± 0.03 mg/l as lethal low O_2 concentrations for larval carp hatched in summer, and 0.99 ± 0.02 mg/l for those hatched in autumn and winter. Using a decrease in O_2 consumption as an index to O_2 toxicity, Hoffert *et al.*[16] studied toxic response of shrimp (*Penaeus setiferus*), false pilchard (*Harengula chapelaki*), fathead minnow (*Pimephales promelas*), crayfish (*Orconectes propinquus*), fiddler crab (*Uca pugnax*), and mouse (*Mus musculus*) to 5 atm absolute of 100 percent O_2. The two freshwater species (minnow and crayfish) were able to tolerate these conditions without a decrease in O_2 consumption, while the saltwater species responded with significant decreases in consumption. It was postulated that because of the relatively stable O_2 concentrations in their environment, marine forms had not developed a mechanism to deal with the fluctuation in O_2 common to freshwater environments. Courtot [17] studied the respiration of the longjaw mud sucker (*Gillichthys mirabilis*), a fish inhabiting both fresh and salt water. O_2 consumption increased directly with temperature of acclimation, inversely with available O_2 in the water, rose at salinities from 14 to 42 g/l (ppt), and fell again at salinities from 42 to 70 g/l. These physiological adjustments to varying environmental conditions were discussed in relation to the animal's wide range of habitats.

Weintraub [18] studied heart rates of free-swimming rainbow trout at different O_2 concentrations by means of radio-telemetry. Under normoxic O_2 concentrations (150 mm Hg), heart rate was directly related to normal activity patterns of individual fish. Onset of bradycardia (slowed heart rate) occurred at higher O_2 concentrations among less active individuals and more frequently than among active individuals. Hough [19] studied blood changes in mudfish (*Umbra umbratus*) acclimated at 8° or 25°C and held at either high (60 to 72) or low (45 to 50) percent O_2 saturation at each temperature. Acclimation to high temperature involved increases in hematocrit and hemoglobin values, but acclimation to different O_2 concentrations at a common temperature involved changes in the amount of hemoglobin present in recirculating blood. Energy consumption in 3-year old goldfish (*Carassius auratus*) held at 20°C in a sealed container without addition of fresh water was studied by van den Thillart *et al.*[20] Anoxic conditions were produced for several hours. Dorsal white muscle (having much higher ATP and GTP than whole fish) was the study tissue. Muscle ATP decreased markedly among 15-hour anoxic fish. A comparable rise in inosine-monophosphate suggested that it was the end product into which all adenosine compounds were converted. Balancing of energy production and consumption processes and products were discussed, with the conclusion that, during anoxia, energy produced from O_2 reserved within the goldfish, phosphorylated compounds and glycolysis, can only cover one-third of the energy need. Bronchart-Lannoye [21] described the influence of several environmental factors on the extent of branchial and air breathing in a number of fishes. The factors studied were: concentration of O_2 in the water, relationship between branchial and aerial breathing, the influence of light period on the frequency of aerial breathing, the influence of anoxic water, and the effect of aerial anoxia. Results showed that branchial and aerial breathing systems functioned with varying degrees of independence from one species to another.

Curvature of the peduncle and absence of caudal fins were reported by Rucker [22] among chinook salmon (*Oncorhynchus tshawytscha*) fingerlings believed exposed to low O_2 during embryogenesis. Inadvertent flow changes during incubation resulted in death of 20 percent of the embryos from apparent suffocation. Supersaturation of dissolved gases did not contribute to coagulated yolk disease among fry initially exposed as embryos to 103 percent O_2 and 103 percent N_2. Adair and Hains [23] com-

pared the degree of N_2 and its supersaturation at three power stations on Lake Norman, N. C., and found that the change in temperature of the discharge water over intake was an important factor contributing to percent saturation and that the depth of discharge was also important. These two factors (together with the volume of discharge) had a direct relationship to the extent of gas bubble disease (GBD) among associated fishes. Nebeker *et al.*[37] found that time to death among juvenile sockeye salmon (*Oncorhynchus nerka*) exposed to air supersaturated water was most affected by total gas supersaturation. No mortalities occurred at concentrations below 100 percent regardless of O_2/N_2 ratios. The CO_2 concentration (1.7 to 22.0 mg/l) had no significant effect on the time to death when the percent saturation of total gases was held constant. Much more extensive and severe signs of GBD developed at high O_2/N_2 ratios. Mortality was lower when the same percent total gas supersaturation resulted from N_2 concentrations near 100 percent and the O_2 concentration elevated than in the converse. Rucker[38] studied the effects of different O_2/N_2 ratios on the survival of juvenile coho salmon (*Oncorhynchus kisutch*) of different sizes. Holding the total dissolved gases (TDG) constant at 119 to 131 percent supersaturation and the temperature at 15.6°C, smaller fish (3.8 to 6 cm) were more resistant than larger fish (8 to 10 cm). The time to death at 120 percent saturation of combinations of N_2 and O_2 concentration became abruptly less in going from 109 percent N_2:150 percent O_2 to 105 percent N_2:173 percent O_2. These results suggested that, at 120 percent saturation, N_2 was more important than O_2 in determining toxicity.

Dawley *et al.*[17] found that juvenile fall chinook salmon (48 mm) were more resistant to air-supersaturated water than juvenile steelhead trout (180 mm). At 120 percent saturation, the salmon incurred 50 percent mortality in 32 days, the trout in 30 hours. GBD was evident at 110 percent saturation among fish with available water depths to 3.3 m, and at 105 percent among fish with only 0.23 m available depths. Mean depth distribution of fish in the deep tank increased with the degree of air supersaturation. After return to control water, fish previously exposed to potentially lethal levels of supersaturation recovered in time. Bouck *et al.*[16] studied the effects of several levels of air supersaturation on a variety of fishes, life stages, and physiological responses. Exophthalmia was common at 120 percent saturation and rare at 105 percent, as death at 105 percent occurred more rapidly. Secondary fungal infections and blindness leading to potential starvation and failure to spawn were reported related to induced lesions. Differences in response caused by sex were related to spawning state. When fish were fully acclimated, warmer water tended to reduce the toxic effects. Previous exposures to supersaturation tensions failed to prevent successful transition of smolts from fresh to salt water, or viability of sex products derived from exposed adults. Great differences between species were found, with trout and salmon being the most susceptible of the species studied, and bullheads (*Ictalurus* sp.), largemouth bass (*Micropterus salmoides*), and carp being the least sensitive.

Bouck *et al.*[16] reported GBD among Columbia River fishes taken below power dams. There was no evidence of GBD in lampreys, but incidences of 40 and 20 percent were observed in the salmonids and American shad (*Alosa sapidissima*), respectively. Fish damage was not noted during the 1973 low water year when spillage over the dams, the primary cause of gas supersaturation, was essentially eliminated. Schneider *et al.*[18] ranked nine fish species in order of increasing tolerance to gas supersaturated water: mountain whitefish (*Prosopium williamsoni*) < rainbow trout < large scale sucker (*Catostomus macrocheilus*) < channel catfish (*Ictalurus punctatus*) < black bullhead (*Ictalurus melas*) < bluegill (*Lepomis macrochirus*) < pumpkinseed sunfish (*Lepomis gibbosus*) < smallmouth bass (*Micropterus dolomieu*) < carp (the most tolerant). When bullheads were exposed to gas supersaturated water after acclimation to 8°, 15°, and 30°C, those at 8°C were more tolerant to supersaturated conditions than those at higher temperatures. Dawley and Ebel[17] studied the effects of supersaturation of dissolved N_2 + Ar on juvenile chinook salmon and steelhead trout. Adverse effects were found influenced by TDG, as the same percent supersaturation of N_2 + Ar was more toxic when higher test levels raised the TDG from 110 to 113 percent. Toxic effects were found at TDG between 110 and 115 percent. Mortality was the principal factor studied, but reductions in growth, swimming ability, and blood calcium and increases in deformities were also found. Six- to 8-week-old channel catfish, held 1 week in water with vigorous aeration, developed gas emboli in the peritoneal cavity and 85 percent died.[17]

The relative resistance of juvenile chinook salmon from different natal areas to air super-

saturated water was studied by Cramer and McIntyre.[12] Fish in the Trask River, where lethal air supersaturation has never been observed, were less tolerant of air supersaturation than Columbia River fish, and upper Columbia River stocks were more tolerant than lower Columbia River stocks. However, heritability was low, indicating that no great increases in resistance can be expected, even at relatively high selection intensities.

Eddy[13] described the responses of rainbow trout to acid stresses caused by infusion of acid directly into the blood stream or by increasing the CO_2 concentration of the test water. Introduction of acid into the blood stream resulted in transient increased blood pH and decreased pCO_2 and bicarbonate, while increases in CO_2 in the external medium resulted in increased blood pH and increased pCO_2 and bicarbonate. Ventilation rate and volume increased with both types of acid stress. Blood pO_2 was little changed under both types of stress, but O_2 content was decreased and tended to stay decreased for several hours. Janssen and Randall[14] described the increases in ventilation volume and in blood pCO_2 and pH of rainbow trout in response to increased environmental CO_2 and discussed the changes with respect to the hypothesis of regulation of blood pH in fish via HCO_3 /Cl exchange across the gills.

Romanenko[15] found that fish of the minnow family (Cyprinidae) were capable of enduring wide CO_2 ranges in the environment without undue carbonic acid acidosis. This capacity was believed to result from their ability to maintain blood acid-base balance through bicarbonate buffering. Borjesson and Hoglund[16] studied the gradual adjustment in juvenile Atlantic salmon (*Salmo salar*) swimbladder O_2 and CO_2 content and simultaneous hemoglobin O_2 carrying capacities as influenced by sudden increases in ambient water CO_2 tension. Within about 8 hours after the CO_2 rise in the water, the swimbladder concentration rose from 0.04 percent to 5.5 percent and stabilized. The O_2 concentration decreased from a mean of 14.0 percent to 8.5 percent in approximately 1 hour. The Root effect was found greatly reduced after 80 hours of acclimation to high ambient CO_2. This adjustment was accompanied by a corresponding increase in the blood bicarbonate content. Borjesson and Hoglund[16] also reported that hypercapnia induced elevated muscle and blood lactate levels in Atlantic salmon parr. Muscle lactate increases, from 1.01 ± 0.59 to 2.34 ± 0.65 mmol/kg (wet weight), were statistically significant, while blood lactate changes from 1.09 ± 0.42 to 1.71 ± 1.50 mmol/l were not. Hattingh[17] studied the influence of increased environmental CO_2 on blood glucose levels, hematocrit, and hemoglobin levels in the South African freshwater fish *Tilapia rendalli* and found that the hematocrit and hemoglobin levels fell in direct relationship to the pH change with addition of CO_2, while the blood glucose rose. Hypoxia and hypercapnia should thus be considered as initial causes of "asphyxiation hyperglycemia."

Salinity. Leggett and O'Boyle[18] forced American shad (*Alosa sapidissima*) in floating cages from salt water to fresh water and studied changes of the osmotically active blood constituents. Blood Na and Cl levels decreased in the shad compared to controls tested in salt water, while Ca, glucose, and lactic acid levels increased. Potassium and Mg levels remained stable. High mortality also occurred in the shad rapidly transferred to fresh water. It was suggested that the meandering behavior of migrating shad at the salt-fresh water interface may be a behavioral response to slow the transition to fresh water. The ability of rainbow trout (*Salmo gairdneri*) to acclimate to sea water was evaluated by Landless.[19] Mortality occurred above 24.2 g/l when salinity was gradually increased in tests using a partial renewal recirculating system. Similar results occurred when fish were transferred directly from fresh water to sea cages at 32 g/l salinity, at which 1 to 8 percent died. Whitfield and Blaber[20] determined the salinity and temperature tolerance of *Tilapia rendalli* and related the geographical distribution of the species to the results. The upper salinity limit was 19 g/l between 20° and 28°C. Blood osmotic concentrations ranged from 255 m osmol/l in fresh water to 340 m osmol/l at a salinity of 19 g/l. Tay and Corride[21] studied developmental rates, viability, and hatching success of mummichog (*Fundulus heteroclitus*) embryos incubated at constant levels of salinity and temperature from 0 to 60 g/l and 15° to 30°C. Response surface analysis indicated optimal conditions for embryo survival at 19 to 30 g/l salinity and 18°C. Higher temperatures in all instances accelerated development. The highest salinity (60 g/l) retarded development, onset of hatching, and median hatching time. Embryos hatched with difficulty at the lowest salinity and temperature, 0 g/l and 15°C. The salinity tolerance of California killifish (*Fundulus parvipinnis*) raised at salinities from fresh water to 70 g/l was studied by Rao[22]

in relation to age and incubation salinities. Larvae hatched in lower incubation salinities exhibited greater freshwater tolerance, whereas those hatched at higher salinities exhibited greater hypersalinity tolerance. Freshwater tolerance decreased with age. Yolk sac larvae were completely freshwater tolerant, while 15-day larvae were least resistant. Shaw *et al.*[52] studied the growth of Atlantic salmon (*Salmo salar*) parr receiving daily rations of 0 to 2.0 percent of dry body weight at environmental salinities of 0.1, 25, and 50 percent seawater. Instantaneous growth and food conversion efficiencies were similar at each salinity, while the maintenance ration was slightly higher in 50 percent salinity.

Rashamukhiden and Parvatheswararao[53] studied the time course of adaptation to osmotic stress (increased salinity) in *Tilapia mossambica* using O_2 consumption rates. Adaptation consisted of two phases, an initial rapid rise and a gradual decline to a post-stress stabilized O_2 consumption rate. The level of initial rise and the time to a stabilized O_2 consumption rate were related to ambient salinity but not directly, with both being less for smaller fish. The blood O_2 capacities of American eels (*Anguilla rostrata*) acclimated to three environmental salinities was determined by Guernsey and Polukowich.[54] The eels showed a relationship between O_2 capacity and hematocrit which appeared to be altered by ambient salinity. The brackish-water-acclimated group exhibited the highest mean O_2 capacity and weight-specific O_2 capacity, while hematocrits were greatest in the freshwater group. In addition, Isaia and Hirano[55] studied net water fluxes in isolated gills of freshwater- and seawater-adapted eels (*Anguilla anguilla*) incubated in fresh water and sea water. The results indicated that adaptation to a given medium decreases osmotic permeability, so that water gain in fresh water or water loss in sea water is minimal.

The hematological characteristics of laboratory-acclimated striped bass (*Morone saxatilis*) under four different salinity and temperature regimes were examined by Courtois.[56] Salt water-acclimated striped bass had significantly lower serum K, higher Na, and lower hematocrit and hemoglobin than freshwater-adapted bass. Increased temperatures appeared to cause reverse effects in freshwater- and seawater-acclimated striped bass. Dharamaba *et al.*[57] compared Na and Cl influx and efflux, Mg and Na-K-activated gill ATPase, and transbranchial electric potential in *Tilapia mossambica* adapted to fresh water, one-third sea water, and sea water. Plasma Na and Cl concentrations remained practically constant in all three media. Sodium and Cl effluxes were 10 and 200 times higher in *Tilapia* adapted to one-third sea water and sea water, as compared to fresh water. The Mg- and Na-K-activated ATPases showed parallel increases in activity with increased external salinity. Handling stress caused Na and Cl effluxes 3 to 5 times higher in stressed than in unstressed fish. The effects of changes in salinity and the effects of hypophysectomy on plasma Na and Cl and urine osmolality of channel catfish (*Ictalurus punctatus*) at 23° and 30°C were studied by Norton.[58] A model was proposed defining the limits of osmoregulatory capabilities of channel catfish. Davis and Simco[59] also used the channel catfish to determine the effects of saltwater concentrations, temperature, season, and exogenous cortisol on plasma electrolytes. Plasma Na and Cl were consistently elevated at 10 and 15 g NaCl/l. Cortisol injections had no effect on the increased plasma Cl, osmotic pressure or percent protein of fish transferred from fresh water to 12 g NaCl/l. No effect of season on plasma electrolyte response to salt water was observed, although one experiment suggested that low temperature may limit saltwater tolerance of channel catfish. Rainbow trout were transferred from fresh water to sea water by Milne and Randall.[60] No significant changes were observed in plasma Cl and HCO_3 concentrations, arterial blood pCO_2 and pH, and CO_2 excretion. Results were discussed in relation to various models proposed for regulation of pH in goldfish (*Carassius auratus*) and coho salmon (*Oncorhynchus kisutch*). Lahlou *et al.*[61] evaluated the effects on plasma electrolytes, gill fluxes, and intestinal transport of sodium in seawater-adapted trout (*Salmo trutta*). Plasma electrolyte concentrations varied only slightly from those in fresh water in completely adapted trout. Gill permeability to Na ion decreased. Disturbance of the animals caused disequilibrium and fish failed to adapt to hypertonic media. Adams *et al.*[62] evaluated the temperature tolerance of steelhead (*Salmo gairdneri*) smolts using gill Na-K-ATPase activity and survival in 35 g/l sea water. Maximum ATPase activity and 100 percent seawater survival occurred with smolts reared at 11.3°C and tested in mid-April, and smolts reared at 6°C and tested in early May. The results were related to direct temperature control of metamorphosis and successful migration of juvenile steelhead trout. Ultrastructural changes in the secretory process in caudal

neurosecretory cells of brook trout (*Salvelinus fontinalis*) exposed to fresh water, de-ionized water, and sea water were studied by Chevalier.[98] The study provided further evidence of the involvement of the caudal neurosecretory system in osmoregulation in fish. Preference experiments on elvers and young eels were conducted by Schulz,[97] and elvers preferred fresh water to water of 10 g/l and 30 g/l salinity. Young eels preferred water of 10 g/l salinity. Preferences were not influenced by previous maintenance at different salinities.

pH. Populations of two species of fish were observed by Bosse[49] in acid sections of the South Fork of the Saline River affected by surface mine waste compared to apparently normal populations of fish in the unaffected sections. The number of species and density of macrobenthic organisms was also reduced in the acid sections. Ragsdale and Bulow[93] studied the effect of acid mine drainage on the water quality and fish populations of Dale Hollow Reservoir on the border of Kentucky and Tennessee. Based on creel census data, lower productivity was indicated in areas affected by acid mine drainage. The food habits of the brown bullhead (*Ictalurus nebulosus*) were investigated by Kharberg and Benson[100] in an acid-mine polluted river and in an acid-free impoundment in West Virginia. Food habits were related to availability, although some selectivity for chironomids occurred. Brown bullheads consumed detritus, wastewater, and acid-tolerant invertebrates in the polluted river. The brown bullhead was also studied by Fowler[101] in relation to its fecundity in the acid-mine-polluted Monongahela River, West Virginia. The fecundities matched or exceeded the majority of values previously reported for the species. Harvey[102] compared the fish communities in a group of acid-stressed Lakes in the La Cloche Mountains, Canada, and demonstrated declining sport fish populations. The number of fish species present and diversity correlated strongly with pH. The cause of acidification of the lake was suggested as acid precipitation originating from the operation of copper smelters in the area. Beamish *et al.*[103] described and documented the effects of acidification at an annual rate of 0.13 pH units on the fish population of George Lake, Ontario, Canada. In response to decreasing pH near or below 5, walleye (*Stizostedion vitreum vitreum*), lake trout (*Salvelinus namaycush*), smallmouth bass (*Micropterus dolomieui*), and burbot (*Lota lota*) disappeared. Brown bullheads, rock bass (*Ambloplites rupestris*), pumpkinseed sunfish (*Lepomis gibbosus*), northern pike (*Esox lucius*), and white sucker (*Catostomus commersoni*) failed to spawn. Additionally, at this pH white sucker showed changes in blood calcium, spinal deformities, changes in size of age classes, and reduction in population size. Blood NaCl levels were reported by Leivestad and Muniz[104] for brown trout (*Salmo trutta*) in stress after a fish kill in the Tovdal River, Norway. The kill followed melting of acid-precipitation-contaminated snow. Trout from the areas with severe fish kills showed lower Cl levels than those from unstressed localities. Stressed fish transferred to water neutralized to pH 6 regained normal NaCl levels after 3 weeks. Values as low at pH 4.0 to 4.8 were measured during the early phase of snow melt.

Huckabee *et al.*[105] working in the Great Smoky Mountains National Park, placed brook trout (*Salvelinus fontinalis*) fingerlings and shovelnose salamander (*Leurognathus marmoratus*) larvae in cages in streams above and below road fills and natural formations containing iron sulfide rock. The bioassay and stream surveys indicated trout could not tolerate the stream conditions below the acid rock. The pH in the stream ranged from 4.5 to 5.9. Chemical analyses also indicated increased sulfate and heavy metal concentrations below the exposed acid rock. The low pH tolerance of brook trout was related to weight, temperature, season, and strain, according to Robinson *et al.*[106] Survival times were directly related to weight and inversely related to temperature. Brook trout tested at pH 3.35 to 3.50 had maximum survival times in the summer. Tests of different strains indicated pH tolerance was heritable and that brook trout strains with lower pH tolerances can likely be developed by selective breeding. Menendez[107] measured the chronic effects of continued exposure to reduced pH on adult and developmental stages of brook trout. In 5-month exposures of adults, significant reductions occurred in survival, growth, and number of viable eggs at pH levels of 4.5, 4.8, and 5.0, respectively. Significant reductions in hatchability of embryos from exposed and unexposed parents occurred below pH 6.5, and growth and survival of alevins was reduced at pH levels below the control (pH 7.0). Johansson and Milbrink[108] and Milbrink and Johansson[109] incubated fertilized eggs of the perch (*Perca fluviatilus*) and the roach (*Rutilus rutilus*) at different pH values in laboratory and field experiments. In the laboratory, both species showed a 50 percent decrease in

hatching at a pH of 5.0 and an almost complete lack of hatching at 4.0. The field experiments indicated that perch eggs were less sensitive to low pH than roach eggs. There was a 93 percent hatching frequency of perch eggs at a pH of 4.7 to 5.0 in Lake Trehörningen where no roach eggs hatched.

The O_2 consumption rate and ventilatory response of rainbow trout (*Salmo gairdneri*) within the pH range of 6 to 9 were investigated by Hargis.[110] Consumption rates at a swimming velocity of 24 cm/s at pH 6 and pH 9 were higher than at neutral pH. Ventilation rates were also higher at pH 6 and 9 than at neutral pH. Coughs (respiratory reversals) were pH-dependent, lower in frequency at pH 6, and higher at pH 9 than at neutral pH. Pegg and Jenkins[111] measured the O_2 consumption rates of bluegills (*Lepomis macrochirus*), pumpkinseed sunfish, and brown bullheads exposed to acid water in a flow-through respirometer. Bluegills and bullheads exhibited decreased mean O_2 consumption rates, while pumpkinseed sunfish showed increased rates in acid water. Ventilation rates for all species in acid water were highly variable and frequently increased to three times the standard resting rate.

PESTICIDE POLLUTANTS

Insecticides. Hamelink and Waybrant[112] found that DDE added to a flooded quarry distributed rapidly throughout the system, whereas, lindane persisted in the epilimnion until turnover. Concentration factors for zooplankton at equilibrium averaged 33 for lindane and 4.5×10^5 for DDE, while the same values for bluegill (*Lepomis macrochirus*) were 768 and 1.1×10^5, respectively. Rainbow trout (*Salmo gairdneri*) achieved a concentration factor of 1.8×10^5 for DDE in 108 days. Boelens[113] determined that warm- and cold-water fish contained 3.0×10^5 and 3.6×10^5 more DDT than the water in a Canadian lake. Death of lake trout (*Salvelinus namaycush*) fry was attributed to DDT. After adding ^{14}C-DDT to a small pond, distribution between water, sediment, and a wide variety of aquatic organisms was studied by Salonen and Vaajakorpi.[114] Mesenteric adipose tissue of perch (*Perca fluviatilis*) contained 23.8 µg/g. Lower concentrations were found in carp (*Cyprinus carpio*). Craig and Rudd[115] demonstrated that a linear, first-order model adequately represented the distribution and fate of DDT-type compounds in the Clear Lake ecosystem. Studies of residues found in large pelagic and benthic fish were included. Up to 10 µg g DDT-type compounds, but no dieldrin or toxaphene, were found in muscle from wild fish in Sudan by El-Zorgani.[116]

No consistent relationship was observed by Bulkley *et al.*[117] between dieldrin concentrations in muscle tissues of channel catfish (*Ictalurus punctatus*) from the Des Moines River, Iowa, and length, age, sex, or fat content. Kellogg and Bulkley[118] found that dieldrin concentrations in four fish species declined in summer and increased in fall, even though water concentrations declined in both periods. Channel catfish showed a variety of trends, depending on size and year. Deelstra *et al.*[119] determined the level of chlorinated hydrocarbon pesticides in fish collected from Lake Tanganyika in Central Africa during 1971 to 1973. Results from suspected samples of the fish used as a source of protein indicate that the amounts of chlorinated hydrocarbon pesticides present were below the allowable limits set in most countries. Welch *et al.*[120] analyzed wild fish for 1 year after field application of mirex and found a maximum of 2.0 µg/g in mosquitofish (*Gambusia affinis*) 6 months after application.

Fish collected below an effluent from a manufacturer of organophosphate and carbamate pesticides had significantly lower levels of acetylcholinesterase activity than similar fish collected upstream, according to Coppage and Braidech.[121] Low concentrations of the effluent also decreased acetylcholinesterase levels. Bioconcentration factors in mosquitofish in a model ecosystem of 465, 1 143, and 1 324 were found by Lu *et al.*[122] for chlordane, heptachlor, and heptachlor epoxide, respectively. In another model ecosystem, Sanborn *et al.*[123] observed that *Gambusia* contained 8 258 and 4 247 times as much chlordane and toxaphene, respectively, as did the water.

Atchison and Johnson[124] observed that when adult brook trout (*Salvelinus fontinalis*) were fed labeled DDT, 8 percent of the radioactivity in eggs at fertilization was DDD or DDE, and 58 percent in fry 80 days after fertilization was DDD or DDE. By injecting Atlantic salmon (*Salmo salar*) fry, Addison *et al.*[125] found that 40 percent and 90 percent of DDT and aldrin, respectively, were eliminated in 8 weeks. Schaper and Crowder[126] observed a linear uptake by mosquitofish exposed to 3 µg/g toxaphene for 8 hours and no detectible elimination in 6 hours. Methods for determining and characterizing toxaphene in fish and water using polyurethane foam, gel permeation and silicic acid chromatography, and gas chromatography-mass spectroscopy

were developed by Stalling and Huckins.[11] Kobylinski and Livingston[70] found that the bluegill (*Lepomis macrochirus*) accumulated mirex from sediment in both static and flow-through tests. Bioconcentration factors of 1 800 and 10 400 occurred in the static and flow-through tests, respectively, suggesting the possible absorption of mirex from the substrate. Scales and Yarbrough[152] measured endrin in five tissues of both resistant and susceptible mosquitofish exposed to 10 and 1 000 µg/l endrin for 6 hours. Results suggested that resistant fish take up less of the pesticide and are less sensitive at the target site. Brains and livers of susceptible mosquitofish accumulated endrin from a 10 µg/l solution faster than did resistant fish, but at 1 000 µg/l the brains accumulated more and the livers less, according to Fabacher and Chambers.[153] Argyle[154] studied the effect of 0.4, 0.8, and 4.0 µg/l dieldrin in the food for 240 days on the growth of channel catfish and the uptake and elimination of dieldrin. Only at the highest concentration was growth decreased and uptake continuous. Elimination rates were the same at all three concentrations. The existence of a membrane barrier was postulated by Watkins and Yarbrough[155] based on the difference in uptake of aldrin and dieldrin by resistant and susceptible mosquitofish. Reinbold and Metcalf[156] demonstrated that piperonyl butoxide inhibited the metabolism of aldrin, methoxychlor, and trifluralin by green sunfish (*Lepomis cyanellus*). Dale and Miles[157] demonstrated that eluting a column consisting of acetonitrile on Florisil with hexane and acetone completely separates many phosphorus pesticides from fish fat. Solvent extraction, methylation, and gas chromatography (GC) were used by Cook and Moore[158] to measure malathion and its three metabolites in fish tissue. Dale *et al.*[159] measured chlorphoxim in fish by using solvent extraction, column chromatography, and GC with a phosphorus detector. The use of high-pressure liquid chromatography to separate organochlorine compounds from triglycerides was studied by Rohrbeder *et al.*[160] Johnson *et al.*[161] demonstrated that gel permeation chromatography separates a wide range of pesticides from fish lipids. A rapid method for measuring endrin using sequential solvent extraction and thin layer chromatography was developed by Tewari and Sharma.[162] Hollifield[163] identified cholesterol as a potential interference in the analysis of pesticides in fish tissue extracts prepared by gel permeation and Florisil column chromatography.

Mayer *et al.*[164] found that toxaphene adversely affected growth and survival of adult brook trout at 0.008 µg/l and at fry at 0.030 µg/l. Bone collagen may be a sensitive indicator of abnormal growth and development, and have bioconcentration factors up to 70 000. Acute and life cycle toxicity tests were conducted with lindane by Macek *et al.*[112] The TI_m values ranged from 90 to 455 µg/l, whereas the application factors ranged from 0.01 to 0.04 for bluegill, fathead minnow (*Pimephales promelas*), brook trout, water flea (*Daphnia magna*), midge (*Chironomus tentans*), and scud (*Gammarus fasciatus*). Fathead minnows are chronically exposed to aroclor, heptachlor, endosulfan, and trifluralin by Macek *et al.*[165] Incipient TI_m values ranged from 0.60 (endosulfan) to 119 µg/l (trifluralin). Maximum acceptable toxicant concentrations based on successful growth, survival and reproduction were between 11.1 and 41.7, 0.80 and 1.84, 0.20 and 0.40, and 1.0 and 5.1 µg/l for aroclor, heptachlor, endosulfan, and heptachlor, respectively. Residues of trifluralin and heptachlor (including heptachlor epoxide) were determined at the end of the chronic exposures. Bioconcentration was linear over and proportional to the tested concentrations, with observed bioconcentration factors of 1 000 for trifluralin and 30 000 for heptachlor and heptachlor epoxide. Verma *et al.*[166] conducted acute mortality tests on lindane using *Cirrhina fasciata* and studied histopathological changes in selected tissues. The 96-hour TI_m values were 0.04 and 0.41 at 18° and 33°C, respectively. Tissue damage was observed in the gill, liver and kidney. Miura and Takahashi[167] determined the 96-hour TI_m of a synthetic pyrethroid, S-3478TA, to the mosquitofish to be 2.6 µg/l. The effect of hardness, temperature, and decomposition on the toxicity of pyrethrins and pyrethroids to six species of fish in both static and flow-through tests was studied by Marshall *et al.*[168] Coldwater species were more sensitive than warmwater ones. The toxicity of the pyrethroids was not affected by pH in the range of 6.5 to 9.5. The two most toxic pyrethroids were deactivated more rapidly than the other compounds. Macek[169] observed that 11 of 20 combinations of pesticides showed greater than additive toxicity to bluegills, whereas, only one was less than additive. Many organophosphorus insecticides had greater than additive toxicity when combined with other chemicals. Methyl parathion did not significantly affect the acute lethality of endrin to largemouth bass (*Micropterus salmoides*)

fingerlings in tests conducted by Pahapoff.[4] Lunn *et al.*[13] found that carbaryl and dieldrin did not significantly affect respiration, coughing and heart rates of rainbow trout at concentrations below those that caused mortality, but that DDT did. At 10 percent, but not at 1 percent, of the 72-hour TL_m carbaryl, chlordane, parathion, and pentachlorophenol significantly increased the opercular rate of largemouth bass in tests conducted by Morgan.[12] Statham and Lech[14] demonstrated that both azocrotine and carbaryl increased the toxicity of 2,4-D n-butyl ester and Bayer 73 to rainbow trout by increasing the uptake of the compounds from water. Atropine blocked the increase in toxicity and increased uptake. Malathion was more toxic to the eastern mudminnow (*Umbra pygmaea*) than any of its hydrolysis products in both static and flow-through tests, according to Bender and Westman.[15] By studying the effect of guthion on all life stages of the fathead minnow, Adelman *et al.*[16] found that 0.01 µg/l, but not 0.00 µg/l, drastically reduced egg production, but caused no other adverse effects. Unai *et al.*[17] determined the 7-day TL_m of malathion to guppies (*Poecilia reticulata*) to be 0.82 mg/l. Burkovskii *et al.*[18] determined the acute toxicity of chlorophos, DDVP, kytazine, and trichlorometaphos. Maximum allowable concentrations based on sanitary hygienic standards were tests to juvenile carp and sturgeon. Regulations concerning treatment, dilution, or release of wastewater should consider toxicity to fish. Koburicheva[19] compared the toxicity of chlorophos, enterobacterin, and nucleopolyhedrosis virus to trout. Chlorophos disturbed blood circulation of internal organs and caused cerebral plethora and distrophic changes in nerve cells at concentrations of 0.25 to 2.0 mg/l in water. Juvenile trout were more susceptible than adults. Preparations of biological origin did not cause adverse effects and pathological changes were caused only by very high concentrations (0.1 and 1.0 g/l) of enterobacterin.

Svobodova and Studnicka[20] demonstrated that inhibition in carp brain of acetylcholine hydrolase by pesticides was both time- and concentration-dependent and reported that fenitrothion, dichlorvos, and imidan caused inhibition at 60, 81, and 92 percent, respectively. Fenitrothion, dichlorvos, and imidan significantly increased the mean corpuscular volume and packed cell volume at TL_m but decreased the mean corpuscular hemoglobin concentration in carp blood, as reported by Svobodova.[21] Klaverkamp *et al.*[22] observed that rainbow trout fed pellets containing DDT succumbed to and recovered from anesthesia by phenoxyethanol, but not MS-222, faster than control fish. By exposing fathead minnows to DDT in food and water for 200 days, Desaiah *et al.*[23] found that DDT inhibited one ATPase in the brain but stimulated two others. Although a few pesticides stimulated Mg ATPase from brain tissue of bluegills, Yap *et al.*[24] found that all except mirex inhibited Na K-ATPase. Litsharski and Litsharski[25] reported that 0.01 and 0.8 mg toxaphene/l caused 50 percent and 80 percent inhibition of plasma acetyl cholinesterase activity in carp, respectively, but increased the erythrocyte activity by 30 percent and 45 percent, respectively. Monitoring fish for malathion-induced gut and acetylcholinesterase inhibition were considered by Cook *et al.*[26] to be better indicators of malathion poisoning than monitoring fish or water for malathion or malaoxon. Ghosh and Konar[27] showed that 0.5-hour exposure to 10 mg phosphamidon/l inhibited gut invertase activity in catfish (*Clarias batrachus*). By studying the temperature preference of brook trout after exposure to toxicants for 24 hours, Miller and Ogilvie[28] found that 10 and 50 mg DDT/l and 9 mg phenol/l lowered the selected temperature, 50 mg DDT/l raised it, and 25–100 mg PCB/l had no effect. In addition, Ogilvie and Miller[29] demonstrated that exposure to 50 µg DDT/l for 24 hours raised the preferred temperature of Atlantic salmon for 3 weeks. Based on the results of tests with 14 organic and inorganic compounds and Atlantic salmon, Peterson[30] suggested that compounds which are more toxic at higher temperatures caused fish to prefer low temperatures. Rand[31] demonstrated that a 24-hour exposure to parathion affected the reaction of goldfish (*Carassius auratus*), bluegill, and largemouth bass in combinations of flow and odor, both immediately after exposure and 3 weeks later.

Herbicides. Berry[32] determined the effects of a herbicide treatment on the Chickahominy Reservoir, Va. A mixture of diquat dibromide and potassium endothall was applied at 2.5:1 l of each chemical per 0.4 surface hectares. Tissues of bluegills (*Lepomis macrochirus*) collected after treatment revealed no histological effect. Tests in the laboratory showed that concentrations of 11.0 mg diquat/l and 17.0 mg endothall/l, separately or combined, did not cause histopathological changes in bluegills or goldfish (*Carassius auratus*) after 96 hours. Concentrations of diquat (1.1 mg/l) and a mixture (1.1 mg diquat/l and

1.7 mg endothall/l) were avoided by goldfish. The effect of a mixture of 100 μg/l w/w[1] diquat and 100 μg Cu/l on juvenile chinook salmon (*Oncorhynchus tshawytscha*) was observed by Yeo and Decharcz[17] after an application of the mixture to control algal growth in salmon spawning channels. Juvenile salmon were not harmed by the application which was maintained for 3 hours, although algal growth was controlled. Bartley and Hattrup[17] determined the concentrations of acrolein in irrigation water at the Columbia Basin Project, Washington, and the effects of this herbicide on rainbow trout (*Salmo gairdneri*) at the Bureau of Reclamation's Carter Lake, Colorado Aquatic Weed Test Station. Acrolein concentrations of 0.8 and 0.08 mg/l for exposure periods of 4.8 and 48 hours, respectively, were considered to be reasonably safe for trout. A comparison of the effects of grass carp (*Ctenopharyngodon idella*) and the herbicide diquat in vegetated pools containing golden shiners (*Notemigonus crysoleucas*) and bluegills was made by Buck *et al*.[17] Neither grass carp nor an application of 1.83 kg diquat/ha limited the growth or reproduction of either species. However, growth of both species was accelerated following elimination of filamentous algae by grass carp, indicating a redistribution of nutrients formerly stored in the plant cells.

The toxicity of herbicides dinoseb and picloram to cutthroat (*Salmo clarki*) and lake trout (*Salvelinus namaycush*) was assessed by Woodward.[17] The 96-hour TL_m ranged from 48 to 1 400 μg/l for dinoseb, and from 2 030 to 8 660 μg/l for picloram. Lower pH and higher water temperatures generally increased the toxicity of both herbicides. Sensitivity of both species to dinoseb increased with water hardness. Rates of yolk sac absorption and growth of lake trout fry were reduced at 0.5 μg dinoseb/l and 55 μg picloram/l. In a study on the acute toxicity of herbicides to selected aquatic organisms, Tschen-Schluter[17] found that triazine was toxic to the guppy (*Poecilia reticulata*) at a threshold concentration of <10 mg/l. Results with amitrol and the commercial product Azoplant demonstrated possible variations in toxicity between the active substance and the trade product. Marsh *et al*.[17] conducted acute and life cycle toxicity tests with atrazine and six aquatic animals. Maximum acceptable toxicant concentrations ranged from 0.00 to 0.91 mg/l and the TL_m values ranged from 0.78 to 15 mg/l. The toxicity of Benomyl, thiophanate-methyl, and BCM to freshwater organisms was investigated by Canton.[17] Benomyl was the most toxic of the three fungicides to rainbow trout and the guppy, with 48-hour TL_m of 0.46 mg/l and 3.4 mg/l, respectively. Polizzo and Halacka[17] conducted studies on the toxicity of triphenyl stannic acetate, a fungicide to aquatic organisms, and concluded that to avoid detrimental impact upon zooplankton, phytoplankton, fish, and snails, residues of this fungicide in surface waters must not be allowed to exceed the limit of 0.1 mg/l. Bimbo *et al*.[17] found that 1 mg diquat/l increased the cough rate of yellow perch (*Perca flavescens*) in 24 hours.

Morphological changes and survival effects on monkey kidney cells, Girardi heart cells, and rainbow trout gonad cells cultured in vitro and exposed to various herbicides, were observed by Weiss.[17] Dalapon and fenuron caused inhibition of mitotic activity and reduction in cell survival, while 2,4-D stimulated these processes. All diquat treatments caused mitosis to cease within 24 hours and cell survival to decrease sharply. All effects of herbicide treatment (10 to 500 mg/l) were found to occur more rapidly and with greater severity with increased concentration and exposure time.

The avoidance reaction of rainbow trout fry to nine herbicides was determined by Folmar.[17] The lowest concentration of each herbicide avoided was 0.080 (mg $CuSO_4$/l, 0.1 mg xylene and acrolein/l, 1.0 mg dalapon and 2,4-D/l, 1.0 mg TCA/l, and 10.0 mg glyphosphate, Aquatol and diquat/l. Except for $CuSO_4$, acrolein, and xylene, no avoidance was observed at concentrations of the herbicide below those expected to be used or recommended for use in the field.

Piscicides. Phinney[17] conducted a study to determine the rate of repopulation of a section of the North Fork Musselshell River, Montana, by brook trout (*Salvelinus fontinalis*) after treatment with rotenone. Results showed that brook trout from the unaffected areas upstream had completely repopulated the area after 1 year. The primary repopulation was by young-of-the-year trout. An evaluation of the use of Antimycin A for stream reclamation in the Rock River, Wis., was conducted by Baumann.[17] Treatment initially increased the panfish and gamefish catch, but catch per unit effort fell dramatically between the second and third years after treatment. It was concluded that toxicant treatment and restocking alone appear to be ineffective in managing this river.

The uptake, distribution, and elimination

of antimycin in brown bullhead (*Ictalurus nebulosus*) was determined by Schultz and Herman.[10] Radioactive antimycin was readily taken up in bile and tissues of brown bullhead exposed to 0.048 mg Antimycin A/l for 48 hours. Bile contained the most and blood the least. The highest concentration in the muscle was 0.19 μg/g after a 12-hour exposure. Concentrations decreased with time in the muscle, head, skin, and viscera. In addition, concentrations decreased from 0.11 μg/g to 0.05 μg/g when fish were placed in clean water for 96 hours. Bills and Allen[11] and Gilderhus *et al.*[12] analyzed residues of trifluoromethyl 4-nitrophenol (TFM) in fish. Residues of TFM ranged from 0.02 to 5.00 μg/g in fish muscle, with the amount varying with species and exposure conditions. Generally, TFM uptake was lower at high pH and water hardness. Residues decreased to the detection limit (0.01 μg/g) after fish were withdrawn from treatment for 24 hours, with the elimination of TFM being increased slightly with increased temperature.

Dawson *et al.*[13] found TFM to be more effective against larvae of the sea lamprey (*Petromyzon marinus*) than against the embryo and prolarval stages. Larvae of the American brook lamprey (*Lampetra lamottei*) were less sensitive than larval sea lamprey. Temperature had little effect on TFM toxicity, but toxicity was reduced in hard water, especially at high pH, and TFM was significantly more effective against free-swimming larvae than against those in burrows. Piavis and Howell[14] showed that 10 mg TFM/l at 18°C delayed hatching, increased mortality, reduced larval production, and retarded hemoglobin production in later developmental stages of sea lampreys. These findings suggested that the treatment of streams with TFM at customary rates does not effect a complete kill of sea lampreys in all developmental stages. The toxicity of TFM and Bayer 73 to several species of fish was determined by Bills and Marking.[15] Channel catfish (*Ictalurus punctatus*) were the most sensitive to TFM, and brown trout (*Salmo trutta*) to Bayer 73. Both compounds were significantly more toxic at low pH than at high pH. The toxicity of a mixture of these compounds was additive or less than additive to all species and life stages tested. Olson and Marking[16] found that for several species of salmonids green eggs were more resistant than fingerlings to Bayer 73, antimycin, and Noxfish, but were equally sensitive to TFM. Hardness decreased the toxicity of TFM and Bayer 73 to green eggs.

Hunn and Allen[17] determined the effects of acute exposure to TFM on renal excretion of coho salmon (*Oncorhynchus kisutch*). Coho salmon exposed to 0.1 mg/l for 24 hours exhibited an increased output of urine when compared with controls, but the urinary excretion of Na, Ca, Mg, and Cl was not affected. Also, coho smolts more contaminated TFM than free TFM was excreted during this period.

INDUSTRIAL POLLUTANTS

Inorganics. Ujejowska *et al.*[18] conducted a survey to determine Pb content in saltwater fish from the South Baltic Sea and the North Atlantic and in freshwater fish in lakes near Wolsztyn, Poland. Lead levels found in freshwater fish ranged from 0.050 to 0.040 μg/g. The maximum allowable concentration, according to home food legislation in Poland, is 2 μg Pb/g. Northcote *et al.*[19] analyzed trace metal concentrations in fish from the Fraser River downstream of Hope, British Columbia. As, Ni, Cu, and Mo were not detected in any fish. Hg, Cu, Zn, Fe, and Mn were found in detectable concentrations in virtually all fish sampled. Fish from the lowermost, heavily urbanized, industrial regions of the river did not show elevated levels of metals compared to those of the upper, more agricultural regions. The distribution of selected metals in tissue samples of carp (*Cyprinus carpio*) from the Danube River and Danube Canal was determined by Rehwaldt *et al.*[20] The highest concentrations of Cu, Cr, Fe, and Zn were found in the liver and kidneys. Considerable amounts of Cu were also found in the bone tissue, along with high amounts of Sr and La. Peyton[21] studied the input and distribution of aerially deposited heavy metals in an urban watershed in Gary, Indiana. Lead concentrations (in liver and muscle) were found to be significantly higher in largemouth bass (*Micropterus salmoides*) from this system than from a rural pond. The study indicated that contamination by Cd or Pb, primarily through aerial fallout, may present a hazard to aquatic life in this system. The uptake and distribution of Cd and Cr in bluegills (*Lepomis macrochirus*), largemouth bass, and golden shiners (*Notemigonus crysoleucas*) in Palestine Lake near Warsaw, Indiana, was studied by Atchison.[22] No difference was noted in metal residue concentrations among the three species or among the several age classes of bass sampled. Cd and Cr concentrations were approximately 33 and 48 μg/g, respectively, in the liver of bluegills. Abdullah *et al.*[23] analyzed Zn and Mn in the scales of Atlantic

schoon (*Salmo salar*) and brown trout (*Salmo trutta*) in relation to levels of these metals in waters from North Wales, United Kingdom. The results showed significant dependence of Cd levels of both metals in the snake on the levels in the environment. It was suggested that in trout scales there was a minimum concentration of Zn above which there was a direct proportion between environmental concentration and scale concentration.

A survey of Hg in fish from markets, lakes, and creeks of Bombay and Thana, India, was conducted by Tejani and Hebbar.[101] The results revealed [illegible] bones and muscle of *Tilapia mossambique*, *Mugil dussumieri*, and other varieties of fish from Thana and Mumbra creeks have Hg concentrations greater than 0.5 μg/g. The historical changes in Hg concentrations in walleyes (*Stizostedion vitreum*) from Michigan waters were analyzed by Kelly *et al.*[102] Mercury concentrations in walleye (whole fish) were more variable among localities than between recent and historical samples within localities. Walleye in two of seven localities showed no statistically significant increases in the level of Hg contamination over the past 40 years. The effects of the preservation process of museum specimens on the validity of Hg analysis was discussed. Mercury content in fish from three Ohio lakes was determined by Atmore *et al.*[103] Results showed that carp in Pymatuning and Mosquito Lakes contained [illegible] μg/g. Whereas other fish species contained lower levels (approximately 0.1 μg/g). [illegible] species of fish in Fairport Harbor, Lake [illegible] had Hg contents of 0.25–0.35 μg/g. The use of certain fish species as biological magnifiers for inter-lake comparisons of Hg was discussed. Leatherman *et al.*[104] investigated the inter-lake comparisons of Hg was discussed. Leatherman *et al.*[105] investigated the retention of Hg in the muscle of yellow perch (*Perca flavescens*) and rock bass (*Ambloplites rupestris*) from Lake St. Clair after fish were transferred to uncontaminated ponds for 60 months. Total Hg in fillets had declined 53 percent in perch and 50 percent in rock bass; however, the mean weight of fish increased 66 to 183 percent, respectively. All the reduction in Hg concentrations was attributable to dilution by growth, indicating little change in the total burden of Hg in the fillets. Total Hg content in the musculature of fish from dam lakes in Bohemia was reported by Heitmanek *et al.*[106] Highest Hg content was found in pike (*Esox lucius*), followed by capacious carp (*Aspius aspius*), perch (*Perca fluviatilis*), bream (*Abramis brama*), and roach (*Rutilis rutilis*). Mercury levels above 0.50 μg/g were found in fish from some lakes. Significant relationships between total Hg in the musculature and age, and between Hg content and fish body mass were found. Studies of Hg and pesticide residues in freshwater fish from Bavarian lakes and the rivers Danube, Naab, and Altmühl were conducted by Knoppler and Dorn.[107] Mercury values averaging 0.003 μg/g in carp, 0.047 μg/g in trout, and 0.003–0.040 μg/g in muscle tissues of whitefish in lakes and ponds were found. Fish in the Naab and Altmühl rivers contained 0.01 to 0.67 μg/g, with fish from the Danube River containing higher concentrations of 0.06 to 0.47 μg/g. Olsen[108] determined Hg levels in muscle of northern pike 1 and 5 years after the Hg ban in Sweden. Levels of Hg were lower in fish 5 years after the ban than after 1 year. A direct correlation was found between high Hg levels and fish length. Males had higher Hg levels than females. Mercury levels seemed to increase during periods of starvation. Mercury in channel catfish (*Ictalurus punctatus*) and smallmouth bass (*Micropterus dolomieui*) from the Snake River in Idaho was determined by Benson *et al.*[109] Hg concentrations in flesh of older bass found in free flowing waters were higher than in younger fish found in impounded waters. Reversed conditions with respect to age and habitat were found with catfish. Mean levels of Hg in flesh of all cases except catfish in free-flow waters exceeded the FDA recommended level of 0.50 μg/g. Jeyachandran and Rai[110] conducted a preliminary investigation to determine the incidence of Hg in fish from the Mettur reservoir of the Cauvery river system in India. Mercury concentrations in seven species of fish ranged from 0.027 to 0.08 μg/g. The effect of preparation on the Hg content of fish (*Leuciscus rutilus*) and mushrooms from Germany was determined by Scholene and Diehl.[111] Mercury content decreased slightly in fish after steaming and frying. The cooking residue left after steaming was 0.04 to 0.10 μg/g. The uptake and release of Hg from freshwater sediments was studied by Miller,[112] with emphasis on the interaction between Hg and humus. The importance of this process in the overall Hg cycle of freshwater systems was discussed, with particular reference to the problem of high Hg concentrations in fish.

Calamari and Marchetti[113] studied the acute toxicity of Cu and ammonia to rainbow trout (*Salmo gairdneri*) in Lake Orta, Italy. Results were compared to laboratory results pre-

viously published. The toxic effect to the fish was attributed solely to Cu. The 48 hour TL_m for trout in this lake was 0.070 mg/l, compared to a 48-hour TL_m value of 0.080 mg Cu/l calculated in the laboratory. Hampson[102] found that high feeding high stock interest conditions that favor rapid growth and rearing of rainbow trout also can produce extremely low concentrations of un-ionized ammonia which would be completely safe for these fish. Un-ionized ammonia concentration in the water was consistently between 0.001 and 0.1 mg nitrogen/l or between 1/3 000 and 1/3 of the toxic threshold. The toxicity of chlorinated power plant condenser cooling water to fish was studied by Basch and Truchan.[103] The 48-hour total residual chlorine intermittent concentrations lethal to 50 percent of caged brown trout ranged from 0.14 to 0.17 and 0.16 to 0.19 mg/l for fish exposed to two and four 30-minute chlorinations, respectively. The 96-hour TL_m total residual chlorine values of two plants ranged from 0.08 to 0.08 and 0.17 to 0.18 mg/l for fish exposed to three and six 30-minute chlorinations, respectively. Lethal effects of the chlorinated cooling waters were not observed for fathead minnows (*Pimephales promelas*), brown bullheads (*Ictalurus nebulosus*), or other centrarchid species held in many of the discharge channels. Water resources problems and solutions associated with the new Pb belt of southeastern Missouri were analyzed by Hardin *et al.*[104] Data were collected on the quality of air, soils, streams, flora, and fauna. Results of toxicity tests with fish showed that increased cough frequency and opercular rates occurred after the fish were exposed to relatively low concentrations of both milling reagents and heavy metals. Organic milling reagents seemed to be more toxic to those species selected than heavy metals. Ingols[105] found that the cause of rainbow trout kills in the Chattahoochee River-Lake Lanier system, Georgia, and a Greers Ferry Hatchery, Arkansas, were caused by the presence of Mn and H_2S. It was concluded that there may be a synergistic reaction when both are present. Sedlist and Vámos[106] determined that pollution of a lake at Palic, Yugoslavia by H_2S resulted in death of the entire carp population. Wastewater from the industrial town of Subotica caused the pollution, increasing sulphate concentrations from 60 to 800 mg/l. Vámos and Tasnádi[107] showed the alkalization of water by lime ultimately results in the formation of soda and H_2S having unfavorable effects on the development of fish. In general, high concentrations of H_2S caused death, whereas, lower concentrations inhibited metabolism and development. Crippled fish (*Coregonus wartmanni* and *Coregonus macrophthalmus*) from the Bodensee near Bregenz, Germany, were analyzed by Wunder.[108] X-rays showed that spinal forechortening was caused by chemicals from nearby factories. The frequency of fish with deformities has increased from 3 percent in 1964 to 8 percent in 1974.

Buhse and Wach[109] studied the effects of a potash industry on fish in the Upper Weser, Germany. Endopathological changes were evident in fish with S present in the serum and blood smears. Few fish with this condition survived. The effect of Mg, Mn, and Zn ions on the metabolic processes in the liver of carp grown in the cooling reservoir of the Kiev, Ukranian SSR, heating and power plants was studied by Ivanyenko *et al.*[110] In the presence of Mg and Mn salts, growth rate of fish increased, and the aerobic glycolysis and intensity of ^{14}C incorporation, from its inorganic compounds into liver proteins and lipids increased. Effects of some industrial pollutants and factor effluents on kidney peroxidase activity in two freshwater fish (*Ophicephalus punctatus* and *Clarias batrachus*) were recorded by Mukherjee and Bhattacharya.[111] At concentrations of the pollutants at which 70 to 100 percent of the fish survived, peroxidase activity was greatly inhibited, indicating that sublethal doses of these toxicants may cause drastic changes in physiological systems. In some cases, peroxidase activity increased well above the control levels after 8- and 97-hour exposures, but declined toward the end of the test.

Curtis[112] examined the influence of temperature and methylmercuric chloride concentration on the uptake of ^{203}Hg-tagged methylmercuric chloride in bluegills with the use of a verified empirical model. The uptake rate increased with increased temperature and then decreased after a maximum rate had been reached. Uptake rate also increased with increasing methylmercuric chloride concentrations. In addition, uptake of methylmercuric chloride was affected by size, diet, season, disease, and general state of health of the fish. Lu *et al.*[113] studied the fate and effects of Cd and Pb in a laboratory model ecosystem, consisting of algae, *Daphnia magna*, mosquito larvae, snails, and mosquitofish (*Gambusia affinis*). Both metals were readily accumulated by all species, except fish. Fish exposed to 1 percent wastewater sludge accumulated

1.5 μg Cd/g, 130 μg Cu/g, 4 μg Pb/g, and 870 μg Zn/g. The behavior of Cd in a simulated aquatic ecosystem consisting of infusoria snails (*Amphiboria peliobius*), short-bodied catfish (*Corydoras punctatus*), and guppies (*Poecilia reticulata*) was examined by Kinkade.[91] A Cd concentration of 80 mg/l caused death of the test organisms in 24 hours. Uptake and concentration of Cd in comparable tissues from the three species were similar; the gastrointestinal tract (plus contents) accumulated the greatest amount of Cd, progressively lesser amounts were taken up by the kidney, shell (in snails), and gills (in fish). In a related experiment, Kinkade and Erdman[92] investigated the influence of hard versus soft water containing 0.1 mg ^{115m}Cd/l on Cd accumulation in freshwater plants, snails, catfish and guppies. The amount of radioactivity indicated that the initial rate of Cd uptake by these organisms was faster in hard water, but, that the total concentration of Cd was greater in those organisms cultured in soft water. The uptake and effect of Cd on zebrafish (*Brachydanio rerio*) was determined by Rehwoldt and Karimian-Teherani.[93] Cadmium uptake in whole fish was characterized by an initial steep rise, which was followed by slow uptake and finally a plateau. The Cd concentration in females (15.7 μg/g) was approximately double that attained in males (5.1 μg/g). Fish fed 10 μg Cd/g showed marked decreases in the production of offspring as compared to those fed normal fish food. Analysis of offspring showed no detectable amounts of Cd. Smith *et al.*[23] studied the uptake and distribution of Cd in catfish and the effects of Cd on hematological constituents of the fish. Higher Cd concentrations were found in the kidney than in the liver, with concentrations increasing with increasing water concentrations. Biological magnification of Cd decreased with increased water concentrations. Significant differences in hematological constituents were not observed between control and experimental fish. Zinc uptake by Atlantic salmon exposed to lethal concentrations of Zn at 5°, 11°, and 19°C was investigated by Hodson.[94] The rate of Zn uptake increased from a minimum at 5°C to a maximum at 19°C. In fish killed by Zn exposure, Zn concentrations in gills were highest at 19°C and lowest at 5°C and 11°C. Sorensen[95] determined thermal effects on the accumulation of As in green sunfish (*Lepomis cyanellus*). Percent survival decreased and mean As uptake increased slightly as temperature and As concentration increased. Greater As uptake was also observed with increased exposure time. The assimilation and isolation of radioactive sodium arsenate (^{74}As) and ^{74}sodium methanearsonate (MMAA) by algae, daphnids, crayfish, fingerling channel catfish and mosquitofish were analyzed by Woodson *et al.*[97] Bioaccumulation ratios (concentration in tissue/concentration in water) ranged from 4 to 7 for catfish and 137 for mosquitofish. Values declined for catfish after removal from treated water. Distribution of both arsenicals differed with extractant and species, with ^{74}As being found at a higher percentage in the residue than arsenate.

Chernoff[98] developed a faster, safer digestion procedure for analyzing fish tissue for heavy metals using nitric, sulfuric, and perchloric acids. Using atomic absorption spectroscopy (AAS), recoveries were above 98 percent for Cd, Cu, Mn, and Zn at 25, 50, and 100 μg/g. Six different radiochemical methods for the determination of chromium in biological samples were compared by Stella *et al.*[99] The agreement among the data was satisfactory, although elution with KOH/H$_2$O produced the best separation of chromium from protactinium and iridium. Yarbro[100] increased the sensitivity of the diphenylcarbazide colorimetric method for chromium by using a long-path microcell. The results of this method agreed well with those by a flameless atomic absorption method, but an o-tolidine method was apparently subject to interference by vanadium.

The retention and excretion of ^{203}Hg-labelled methylmercury in rainbow trout was monitored by Ruohtula and Miettinen.[101] About 70 to 80 percent of the methylmercury administered to fish in protein-bound form followed a slow excretion rate. An increase in the concentration of protein-bound methyl mercury caused a significant decrease in the biological half-life. Excretion rates were 1.5 times lower at 0.5° in 4°C than at 18° to 19°C. The uptake of methylmercury by aquatic organisms from water and food was evaluated by Lock.[102] The rate of methylmercury uptake by rainbow trout from water was faster than from food, but the percent uptake of Hg was 5 to 10 times higher from the food. Uptake in trout at ≤1.0 μg Hg/l was linearly related to methylmercury concentrations in the medium, with decreases in uptake observed at higher levels. Organic and inorganic Hg in rainbow trout fed with Hg-contaminated diets were studied by Zantiet *et al.*[103] After 8 months, total Hg concentrations in the liver, brain, muscle, gonad, and kidney ranged from 0.88 to 20.8 μg/g. Methylmercury concentrations in the liver, blood, and

kidney ranged from 4.10 to 10.57 μg/g. Trout eliminated up to 13.50 μg Hg/g in the feces. Demethylation of methylmercury by trout was not observed, but a partial methylation of inorganic Hg in the intestine was speculated. Massaro [17] studied the pharmacokinetics of methylmercuric chloride in rainbow trout. After a single intragastric dose (0.5 μg Hg/g) of methylmercuric chloride, maximum concentration factors (CFs) were reached in gills in 1 hour, and in skeletal muscle, brain, and lens after 24, 56, and over 365 days, respectively. Skeletal muscle functioned as a reservoir for methylmercuric chloride and accumulated approximately 50 percent of the dose from 24 to 161 days postadministration. Rate of methylmercury excretion appeared biphasic as a result of slow elimination from skeletal muscle relative to other tissues. Methylmercury also had the ability to rapidly penetrate the red blood cell (RBC) membrane. The erythrocyte transport and transfer of methylmercury to tissues of rainbow trout was studied by Giblin and Massaro.[18] Methylmercury was found to be taken up rapidly and almost completely by RBCs both in vitro and in vivo. The binding of methylmercury to RBC_s was freely reversible in both procedures. Hemoglobin appeared to be the main methylmercury transport protein in trout.

Nishi and Horimoto [19] demonstrated that useful standards could be prepared in proteinaceous material by adding $HgCl_2$ or methylmercuric chloride to gelatin, as long as the concentration of methylmercury does not exceed 2 μg/g. A solvent extraction GC method for determining the concentration of methylmercuric compounds in fish was developed by Schafer *et al.*[20] Mean recoveries for methylmercuric chloride in tissues of three species of fish at 0.2 to 0.6 μg/g levels ranged from 73 to 87 percent, with percent coefficients of variation of 8 to 14. The lower limit of detectability was 0.02 μg/g. Escopeak and Chvojka [21] measured Hg and methylmercury sequentially in fish by flameless AAS and found that the sum agreed well with total Hg as measured by another flameless AAS method. The sensitivity was 0.03 μg, the detection limit was 0.02 μg, and the error did not exceed 10 percent. A method for determination of methylmercury in fish and sediments, using treatment with HBr, solvent extraction and electron capture gas chromatography was published by Krenkel and Burrows.[22]

Birge *et al.*[23] evaluated the use of fish and amphibian eggs as bioindicator organisms to monitor water quality. The frequency of embryonic mortality recorded for 11 monitoring sites correlated with reductions in density and species diversity of pristine populations. Out of four fish species, rainbow trout proved most susceptible to water contaminants. Egg hatchability was markedly reduced in all test waters whose total Hg reached 0.3 μg/l or more. Hutchinson and Uren [24] determined the uptake of $HgCl_2$ by eggs of rice fish (*Oryzias latipes*) and the resulting teratogenic effects. Hemorrhaging, blood vessel deterioration, and loss of blood cells were observed in 70 percent of eggs exposed to 15 μg Hg/l and in all eggs exposed to higher concentrations. Mercury concentrations in eggs exposed to 10 to 30 μg Hg/l for 16 days ranged from 18 to 58 μg/g, respectively. The effects of Hg-contaminated diet upon walleyes were examined by Scherer *et al.*[25] Walleyes fed Hg-contaminated pike contained up to 800 μg/g (wet weight) in the liver and eye lens, with the latter showing the highest average concentrations. Mercury-fed fish developed an emaciated condition and had reduced survival, locomotive activity, coordination, and escape behavior when compared to controls. Quantitative tests demonstrated that Hg-fed fish also had impaired responsiveness to photic stimulation. Spinelli and Stahnken [26] analyzed the effects of diets containing dogfish (*Squalus acanthias*) meal on the Hg content and growth of coho salmon (*Oncorhynchus kisutch*). Results indicated that dogfish meal may be used as a partial (≤50 percent) replacement for the fish meal portion of the diet without encountering Hg values (in the muscle) that exceed 0.5 μg Hg/g. It was observed that growth was decreased in salmon fed Oregon moist pellet type diets in which 50 percent or more of the herring meal was replaced with dogfish meal. The uptake and release of different dissolved Hg compounds by the female guppy (*Poecilia reticulata*) was investigated by Kramer and Neidhart.[27] Accumulation curves showed that the uptake of Hg by fish exposed to methylmercuric chloride was approximately 4 times as fast as those exposed to $Hg(NO_3)_2$. In the presence of EDTA, cysteine, or glutathione, the accumulation rates decreased. The release of inorganic Hg was about 17 times as fast as the release of incorporated methylmercuric chloride. The influence of environmental factors on the uptake and toxicity of several mercurials in fish was examined by Tsai.[28] The high-lipid, soluble members of the mercuric and organomercuric salts were more readily accumulated and more toxic to fish than analogs of low lipid solubility. Both the in-

organic Hg and methylmercury were more readily accumulated by fish from acidic water than from alkaline water. Sodium compounds at low concentrations accelerated the uptake of Hg^{++} but reduced uptake at higher concentrations.

Claspell *et al.*[103] described the use of atomic fluorescence spectrometry in the analysis of fish samples for Hg. Using acid digestion and tin reduction, recoveries of 80 to 90 percent were attained at 1 μg/g, and results correlated well with those obtained by AAS and neutron activation analysis. A strong oxidizing solution for the digestion of fish samples in the determination of total Hg by AAS was developed by Olund and Hackett.[104] Recoveries for mercury and methylmercury were 95 ± 8 and 93 ± 8 percent, respectively. Dugan and Hsardi[105] demonstrated that 1 ng Hg could be rapidly and easily determined with a precision of ±15 percent in biological and aqueous samples by combustion or digestion of samples, collection on a copper micro-column, and determination using flameless AAS. A digestion technique that allows complete recovery of Hg from a wide range of environmental samples, including those containing mercury sulfide was reported by Agemian and Chau.[106] Pearce *et al.*[107] found that digestion in Teflon bombs, Kjeldahl flasks, and borosilicate test tubes gave comparable precisions and Hg recoveries. Experiments with cooked and uncooked fish samples confirmed that no appreciable loss of Hg occurred during baking for 30 minutes at 170°C.

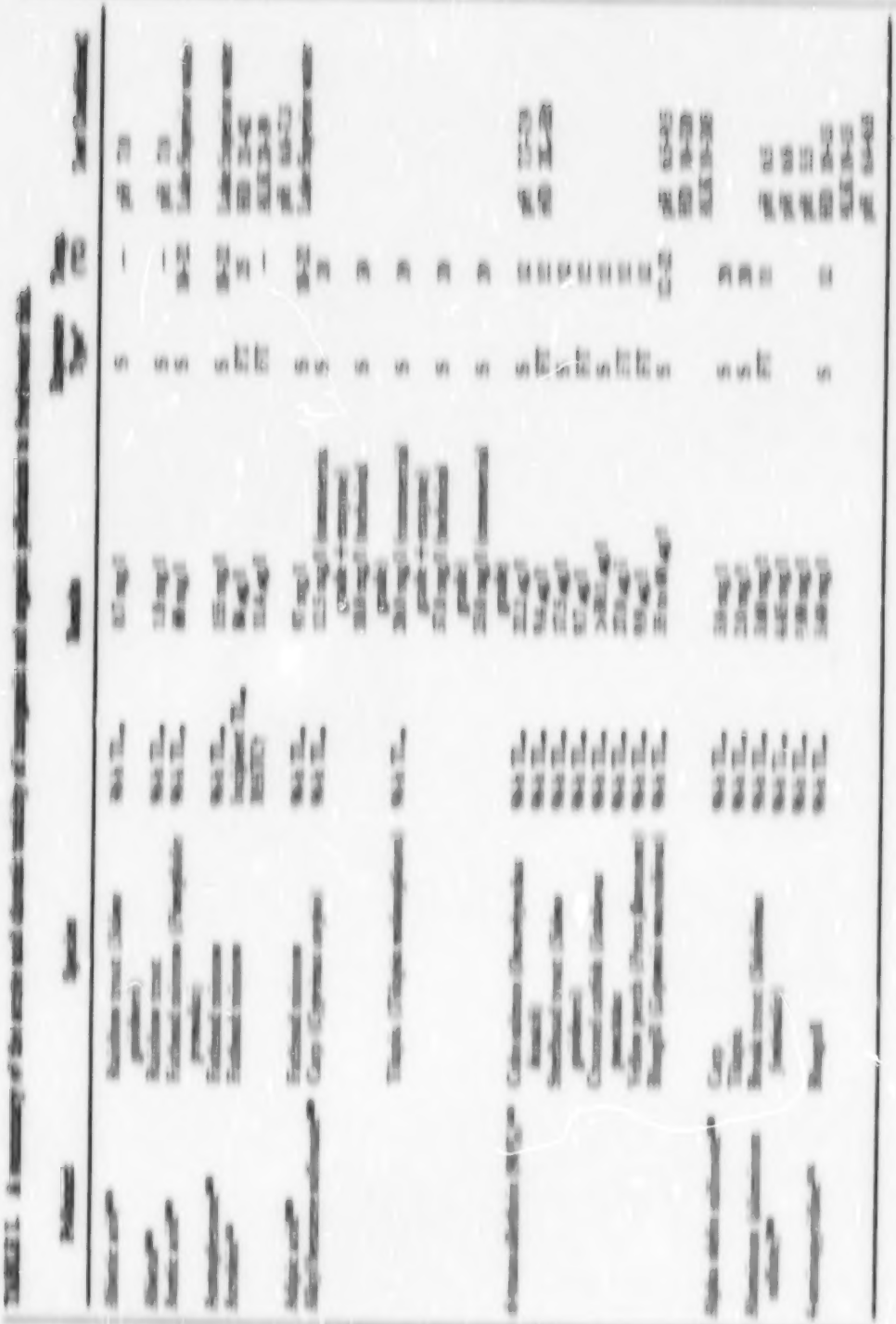
Wobe *et al.*[101] determined the acute toxicity of methylmercuric chloride and $HgCl_2$ to rainbow trout fry and fingerlings. The 96-hour TL_m for fry and fingerlings exposed to methylmercuric chloride was 0.024 and 0.048 mg Hg/l, respectively. The 24-hour TL_m for fingerlings exposed to $HgCl_2$ was 0.90 mg Hg/l. The acute action of both compounds was exerted on the gills resulting in histopathologic alterations. Fingerlings exposed to methylmercuric chloride concentrated Hg in tissue much more rapidly than did those exposed to $HgCl_2$. The effects of methylmercuric chloride on three generations of brook trout (*Salvelinus fontinalis*) were determined by McKim *et al.*[108] The lowest effect concentration of 0.93 μg Hg/l caused deformities in second generation trout and killed all but one female after 108 weeks of exposure. The blood, spleen, and kidney accumulated Hg most rapidly and contained the highest residues in both first and second generation trout, followed by liver, gill, brain, gonad, and muscle in decreasing order. No significant elimination of Hg from tissue of first and second generation fish was observed, although a "steady state" was reached in all tissues after 80 to 88 weeks of exposure. Monomethylmercury made up 90 to 95 percent of the total Hg in muscle, the only tissue analyzed for this compound. Russink and Smith[109] determined the relationship of the 96-hour TL_m to the lethal threshold concentration (LTC) of hexavalent Cr, phenol, and sodium pentachlorophenate for fathead minnows exposed at different temperatures. The LTC's for hexavalent Cr and phenol were less at 25°C than at 15°C, and greater at 25°C than at 15°C for pentachlorophenate. The toxic effects of hexavalent Cr on brook trout and rainbow trout were investigated by Benoit.[110] The 96-hour TL_m values obtained for brook trout and rainbow trout were 59 and 89 mg Cr/l, respectively. Reproduction and embryo hatchability of brook trout were unaffected at Cr concentrations that affected survival of newly hatched alevins. The maximum acceptable toxicant concentration (MATC) for both species exposed to Cr in water with a hardness of 45 mg/l (as $CaCO_3$) was between 0.20 and 0.45 mg/l. The acute and chronic toxicity of Cu to the fathead minnow in water containing a variety of materials from a sewage treatment plant was studied by Brungs *et al.*[111] The 96-hour TL_m values in static tests ranged from 1.6 to 51 mg/l (total Cu) and from 0.60 to 0.98 mg/l (dissolved Cu). The MATC based on survival, growth, reproduction, and egg hatchability in continuous flow tests was between 0.080 and 0.118 mg total Cu/l. The use of dissolved metal concentrations for calculating mortality results and application factors was discussed. Harrison[112] assessed the effects of fish weight, photoperiod, temperature acclimation, and flow rate on the acute lethality of $CuSO_4$ to rainbow trout fingerlings. Median survival time (MST) decreased with increased fish weight, decreased when photoperiod differed from acclimation time, and increased when acclimation time increased. Results of flow rate effects were inconsistent in that the LTT did not increase proportionally with increased flow rate. The toxicity and accumulation of $CuSO_4$ in the stone loach (*Noemacheilus barbatulus*) in hard water was described by Solbe and Cooper.[113] The 33-day TL_m was approximately 0.25 mg Cu/l, with larger fish surviving longer at concentrations greater than 0.20 mg/l. Gill and liver tissue contained the highest Cu concentrations, with the gill, muscle, eye, and vertebral tissue losing significant amounts when fish were placed in

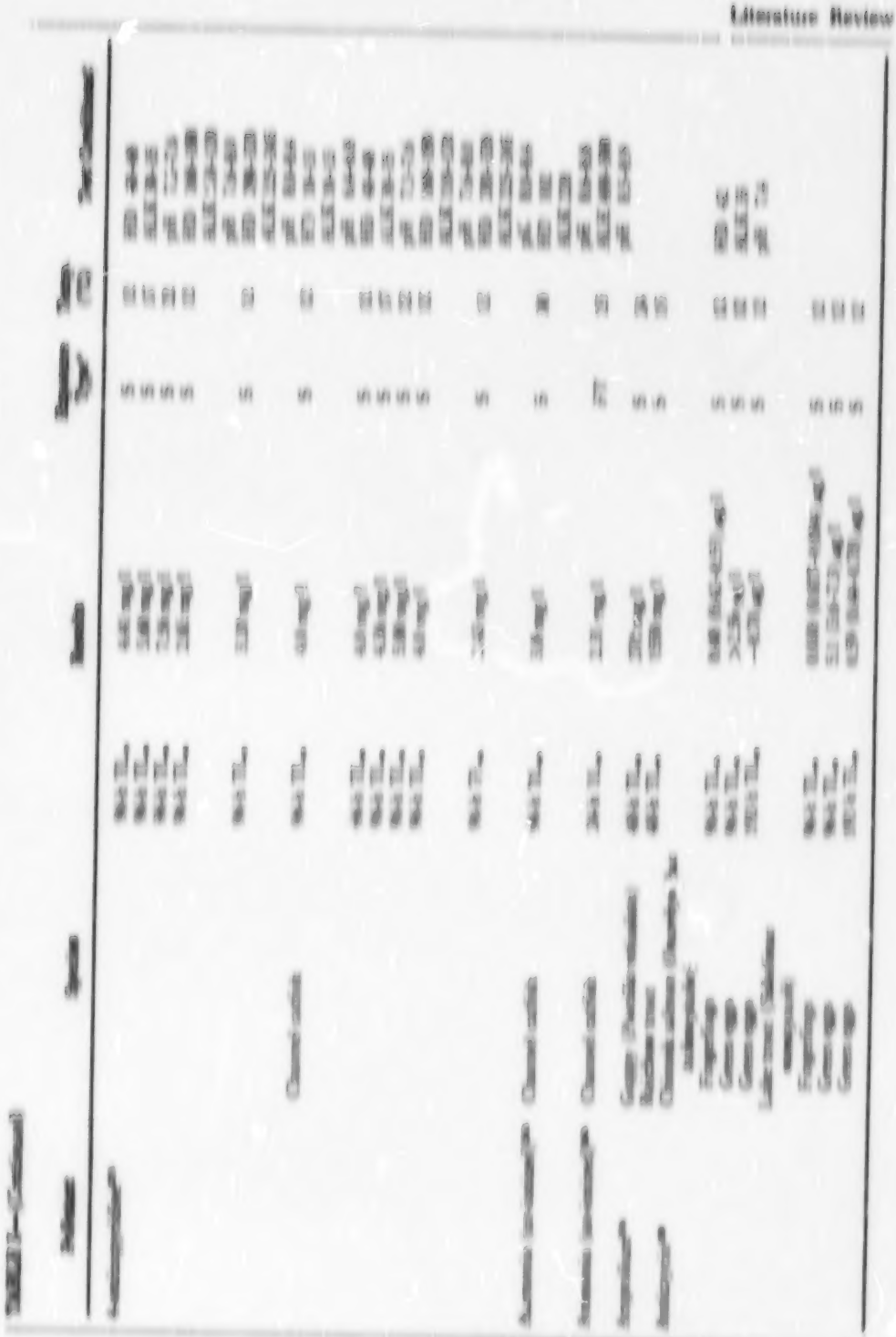
clean water for 7 days. Comparisons of the sensitivity of the stone loach and rainbow trout to Cu, Cd, and Zn were discussed. Hobbsmith *et al*[104] determined the effects of Pb on three generations of brook trout. Results showed that all second generation trout exposed to 255 µg/l and 34 percent of those exposed to 110 µg/l developed scoliosis. Scoliosis also appeared in 40 percent of the newly-hatched third generation alevins exposed to 110 µg/l, and weights of these fish 18 weeks after hatching were also significantly reduced. Gill, liver, and kidney tissue of first and second generation fish accumulated the greatest amount of Pb, with only small amounts accumulated in the edible muscle. Elimination of Pb was up to 70 percent from liver of fish after they were placed in uncontaminated water for 18 weeks. The acute and chronic toxicity of Pb to rainbow trout in hard and soft water was studied by Davies *et al*.[105] The 96-hour TL_m values for total Pb in hard and soft water were 471 to 848 and 1.17 mg/l, respectively. In chronic tests, the MATC values for total Pb in hard water were between 130 and 300 µg/l. In soft water, where exposures were initiated with eyed eggs, the MATC was between 4.1 and 7.8 µg/l, and those initiated with swim-up fry were between 7.8 and 14.6 µg/l. Lautenbacher[106] determined the lethal effects of Pb, nitrilotriacetate (NTA), and Pb-NTA combinations on goldfish (*Carassius auratus*) and developed a method to study the blood, intercellular quantities of Ca and Mg in the blood, and an O_2 depletion measuring system in fish exposed to these compounds. In general, the presence of NTA in Pb test solutions counteracted the lethal effects of Pb. Cd and Zn toxicity to flagfish (*Jordanella floridae*) was determined by Spehar.[107, 108] The 96-hour TL_m values for Cd and Zn to juvenile flagfish were 2 500 and 1 300 µg/l, respectively. In chronic tests, reproduction was the most sensitive indicator of Cd toxicity and was inhibited at 8.1 µg/l. Fish exposed to 1.7 µg/l accumulated up to 78 µg Cd/g after 30 days. In Zn tests, survival of larvae (not exposed as embryos) and growth of females were the most sensitive measures of Zn toxicity and were reduced at 85 and 51 µg/l, respectively. Accumulations of Zn up to 500 µg/g occurred in fish exposed to 47 µg/l after 30 days exposure. The lowest Cd and Zn concentrations causing adverse effects to the flagfish were similar to those affecting other fish species. The effects of Cd on three generations of brook trout (*Salvelinus fontinalis*) were determined by Benoit *et al*.[109] A concentration of 3.4 µg/l caused significant mortality to first and second generation adult males, and retarded growth of juvenile second and third generation offspring. No significant increases in Cd occurred in the edible portions at any concentration. Kidney, liver, and gill tissue accumulated the greatest amounts of Cd, with residues reaching an equilibrium after 20 weeks. Elimination of Cd was rapid in the gill, but no loss was detectable in the liver and kidney after the fish were transferred to control water for 12 weeks. The toxicity and accumulation of As in green sunfish was studied by Sorensen.[110] Median survival times for 100, 500, and 1 000 mg/l were 46, 17, and 13 hours, respectively. Arsenic accumulation increased with exposure concentrations, resulting in values of 314, 541.8, and 980.0 µg/g, respectively. No correlation was observed in comparison of As accumulation with fish length, weight, or condition. Dabrowski[111] studied the effect of As on embryonic development of rainbow trout. Fertile eggs were incubated at 6° to 7°C in sodium arsenate or arsenic trioxide solutions at concentrations of 0.05 to 50.0 mg As/l. The relative toxicity of organic and inorganic compounds of Se to newly-hatched zebrafish was examined by Nimi and Latham.[112] The 96-hour TL_m values ranged from 15 to 50 mg/l for the selenites, and from 81 to 88 mg/l for potassium and sodium selenate. Selenomethionine was the most toxic of all the compounds tested, with the toxicity of selenocystine being similar to the selenates. The probable role of Se in the aquatic environment was also discussed. Cardwell *et al*.[113] determined the acute toxicity of SeO_2 to several freshwater fish species. The decreasing order of species sensitivity was: fathead minnows, flagfish, brook trout, channel catfish, goldfish, and bluegill. Curves relating median lethal concentration to exposure time were characterized by a change in slope indicating a more rapid mortality rate after 96 to 168 hours. The 96-hour TL_m estimates ranged from 2.9 mg/l for fathead minnows to 40 mg/l for bluegills. The acute toxicity of Fe and Al to brook trout was determined by Decker and Menendez.[114] The 96-hour TL_m values for Fe were 1.75 mg/l at pH 7.0, 0.48 mg/l at pH 6.0, and 0.41 mg/l at pH 5.5. The 96-hour TL_m values for Al were 3.6 mg/l at pH 6.5, 4.45 mg/l at pH 6.0, and 4.00 mg/l at pH 5.5. Robinson-Wilson and Seim[115] studied the lethal and sublethal effects of a Zn process effluent (ZPE) on juvenile chinook salmon (*Oncorhynchus tshawytscha*) and coho salmon. The 96-hour TL_m for the ZPE ranged from 3.5 to 36.0 percent by volume and ap-

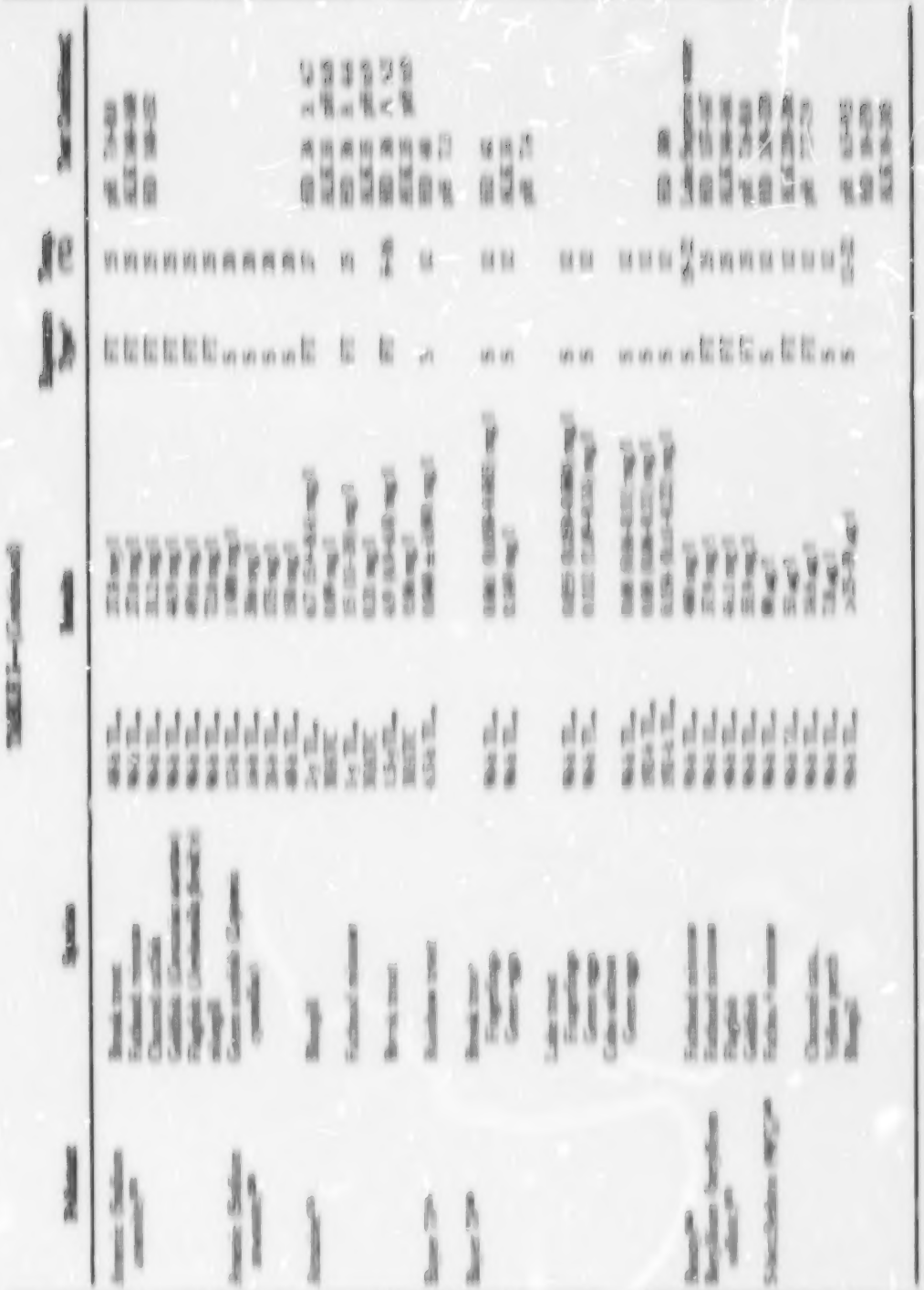
peared to fluctuate with the concentration of ammonia in the EVE. The EPE was more toxic than ammonium chloride solutions in subethal tests measuring growth. The results of the growth studies indicated that EPE could have subethal effects on juvenile salmonids in the river below the outfall. A comparative analysis was made of the effect of different temperatures and concentrations of tin triethyl chloride on eggs and larvae of sturgeon by Danilchenko and Rytina.[116] Significant changes in temperature were shown to cause an adaptive reaction in both eggs and larvae. Lethal and subethal concentrations of the toxicant were found to arrest the development at one of the stages causing death. Lewis[117] evaluated the toxicity of $MnSO_4$ to eggs and fry of rainbow trout. Manganous sulfate appeared to be more toxic to eggs before they reached the eyed stage and at hatching time. Significant difference in mortality of fry exposed to 1, 5, and 10 mg $MnSO_4$/l was not observed within the 72-hour test period. In addition, avoidance of fry to these concentrations was not observed. The toxicity of $KMnO_4$ and its effectiveness for detoxifying antimycin was examined by Markings and Mills.[117] Potassium permanganate (1.0 mg/l) was effective for detoxifying the fish toxicant antimycin in waters of pH 6.5 to 9.5. The 96-hour TL_m for $KMnO_4$ ranged from 0.75 mg/l for channel catfish to 1.90 mg/l for goldfish. The toxicity of $KMnO_4$ was greatest to fish in lower water temperatures, harder water, or in water of higher pH. Cardwell *et al.*[118] determined the acute toxicity of selected toxicants to six species of fish. Estimated TL_m values were: 0.114 mg/l for sodium cyanide, 0.116 mg/l for sodium pentachlorophenate, 2.0 mg/l for selenium dioxide, 18.0 mg/l for sodium arsenite, 93.4 mg/l for beryllium sulfate, and >100 mg/l for lead chloride. Brook trout and fathead minnows were generally most sensitive, followed by channel catfish, flagfish, goldfish, and bluegill. Adelman *et al.*[119] determined the acute toxicity of NaCl, pentachlorophenol, guthion, and hexavalent Cr to fathead minnows and goldfish. The 96-hour TL_m for NaCl were 7,050 and 7,341 mg/l, for pentachlorophenol 0.81 and 0.93 mg/l, for guthion 1.9 and 3.4 mg/l, and for Cr 48 and 130 mg/l for these species, respectively. The experiments demonstrated that the examination of toxicity curves in assessing acute mortality of toxicants allowed comparison between species and toxicants that might not be evident from typical 96-hour tests where TL_m is computed only at 24-hour intervals.

The toxicity of chloramine-containing water to fathead minnows was studied by Capuzzo and Eaton.[120] Results suggested that the toxic action of chlorine compounds to fish was caused by the oxidation of hemoglobin to methemoglobin, producing death by asphyxiation. Bass[121] studied the lethality and toxic mechanisms of intermittent chlorine exposure to bluegills and rainbow trout. Bluegills showed 50 percent mortality in three days when exposed 3 times daily to 0.55 mg chlorine/l. Breathing and coughing rates of rainbow trout increased and blood pO_2, pH, and heart rate decreased during chlorination, recovering only partially after each pulse. Less recovery occurred progressively after each pulse. Based on the bluegill bioassays, the trout physiological studies, and other physiological and histological studies of the exposed fish, it was concluded that the primary mode of action of acute chlorine toxicity was gill damage resulting in death by asphyxiation. Survival time and lethal exposure time for the blacknose dace (*Rhinichthys atratulus*) exposed to free chlorine and chloramines was measured by Tompkins and Tsai.[111] For the concentrations tested, lethal exposure times were found to be shorter than the survival times. The difference between these times decreased with a decrease in total chlorine concentrations. Using either time as a toxicity index, chloramines were more toxic than free chlorine at high concentrations, but less so at low concentrations. Thatcher *et al.*[122] reported the combined effects of chlorine and temperature on juvenile brook trout. Experimental results showed that there was no additional effect of temperature on chlorine toxicity in trout when exposed for 96 hours at 10°C or 15°C. However, 96-hour TL_m values were significantly lower at a test temperature of 20°C, indicating a possible synergistic effect between these two parameters.

Smith *et al.*[110] determined the toxicity of H_2S to various life stages of bluegill. (Acute toxicity to eggs, fry, juveniles, and adults are included in Table 1.) Chronic exposure up to 220 days resulted in no egg deposition at 0.002 2 mg/l and reduced deposition after 97 days at 0.001 mg/l. When exposures were started with eggs, lower H_2S concentrations affected growth than when exposures began with juveniles. Food consumption and time to anesthesia with MS-222 were reduced at 0.000 5 and 0.001 4 mg/l, respectively. The effect of H_2S on fish and invertebrates was studied by Smith and Oseid,[123] Smith *et al.*,[124] and Broderius *et al.*[125] In 150 acute tests, lethal threshold concentrations for juvenile fish







[illegible]—(Continued)

[illegible]	Species	[illegible]	Results	Exposure Type*	Temp. (°C)	Test Conditions†
[illegible]	Eastern redbelly dace (Phoxinus neogaeus)	96-h TL_m	8.5 mg/l	S	20	ALK 38-70
		14-d TL_m	4.2 mg/l	FT	20	pH 7.0-7.3
[illegible]	Channel catfish	96-h TL_m	1 340 µg/l	S	12	HD 388-520
		96-h TL_m	165 µg/l	FT	12	ALK 238-280
	Yellow perch	96-h TL_m	28 µg/l	S	12	pH 7.17-7.9
	Bluegill	96-h TL_m	22.3 µg/l	FT	12	
	Bluegill	96-h TL_m	34-45 µg/l	S	17-22	HD 19-328
						ALK 38-265
						pH 6.5-8.5
2,4-dimethyl phenol*	Rainbow trout	96-h TL_m	3.2-5.8 mg/l	S	9	HD 118-125
						pH 7.3-8.2
Dimethyl phosphorodithioic acid*	Eastern redbelly dace	96-h TL_m	17.8 mg/l	S	20	pH 7.0-7.3
		14-d TL_m	14.3 mg/l	FT	20	ALK 38-70
Dinitrophenol*	Carnivorous trout (Salmo clarki)	96-h TL_m	40-1 330 µg/l	S	10	ALK 48-196
	Lake trout	96-h TL_m	33-1 480 µg/l	S	10	pH 6.5-8.5
Endosulfan*	Fathead minnow	Isozyme TL_m	0.56 µg/l	FT	25	HD 33-42
		MATC	0.28 µg/l	FT	—	ALK 38-39
						pH 6.6-7.3
Endrin*	Longnosed dace (Rhinichthys cataractae)	48-h TL_m	0.27 µg/l	S	20	
Ethion*	Fathead minnow	96-h TL_m	12 600 µg/l	S	18-22	Lake Superior water
Ethylene glycol*	Carp	48-h TL_m (embryo/larvae)	100 mg/l	S	26	
	Rainbow trout	48-h TL_m	23 mg/l	S	10	
Fenitrothion*	Fathead minnow	96-h TL_m	26 mg/l	S	18-22	Lake Superior water
Fenvalerate*	Carp	96-h TL_m	25 mg/l	S	26	
	Rainbow trout	96-h TL_m	>30 mg/l	S	15	
Fluoride*	Fathead minnow	96-h TL_m	32 mg/l	S	18-22	Lake Superior water
Guthion*	Rainbow trout	96-h TL_m	220 µg/l	S	14-16	HD 40.7
						ALK 42.1
						pH 7.4-8.2
Carbofuran	Fathead minnow	96-h TL_m	1 900 µg/l	FT	25	ALK 388
	Catfish	96-h TL_m	2 570 µg/l	FT	25	pH 6.85-7.6
	Fathead minnow	MATC	630 µg/l	FT	23-25	pH 7.3-8.2
Heptachlor*	Fathead Minnow	Isozyme TL_m	7.82 µg/l	FT	25	HD 2.1-42
		MATC	0.86 µg/l	FT	—	ALK 38-39
						pH 6.6-7.3

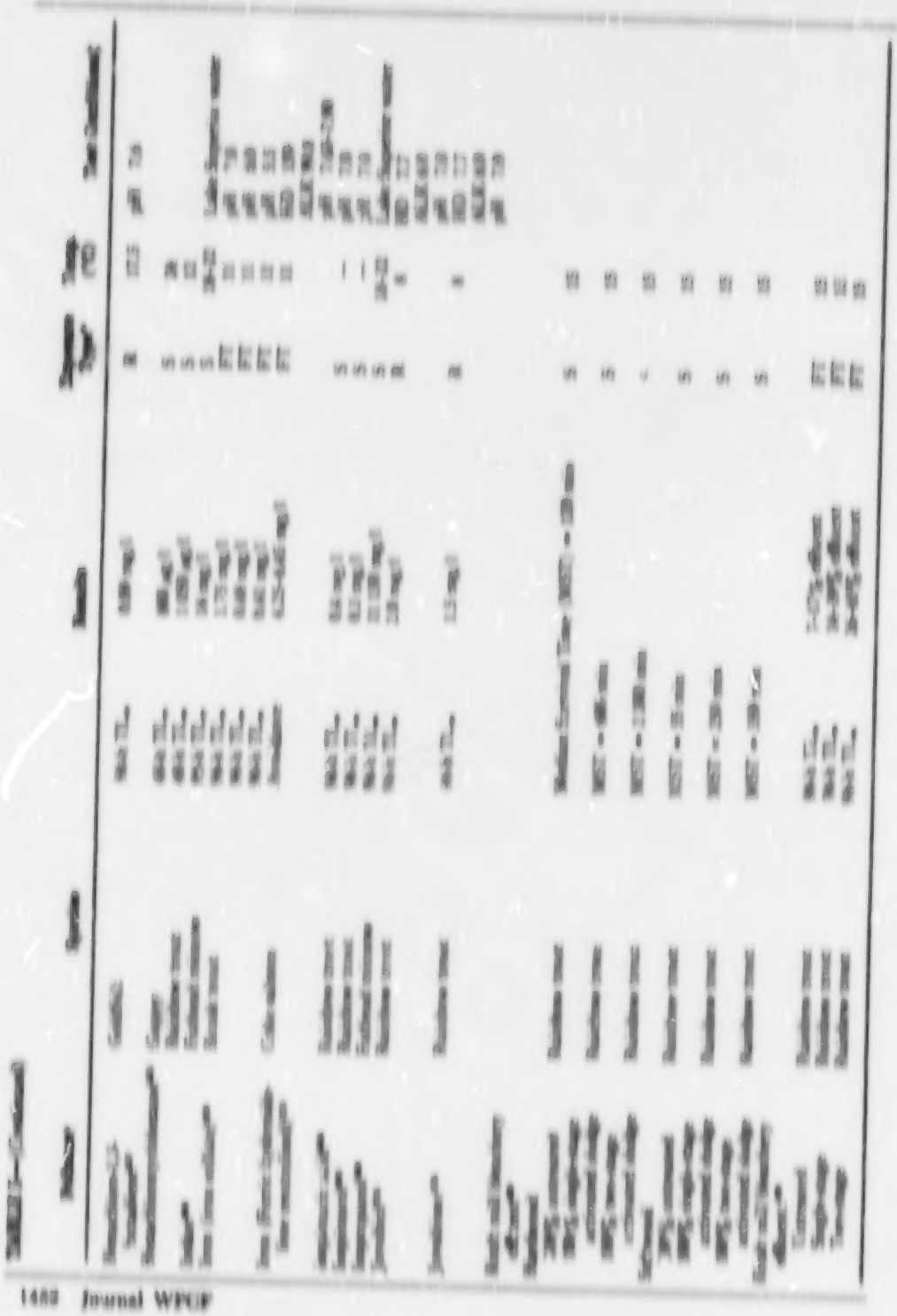
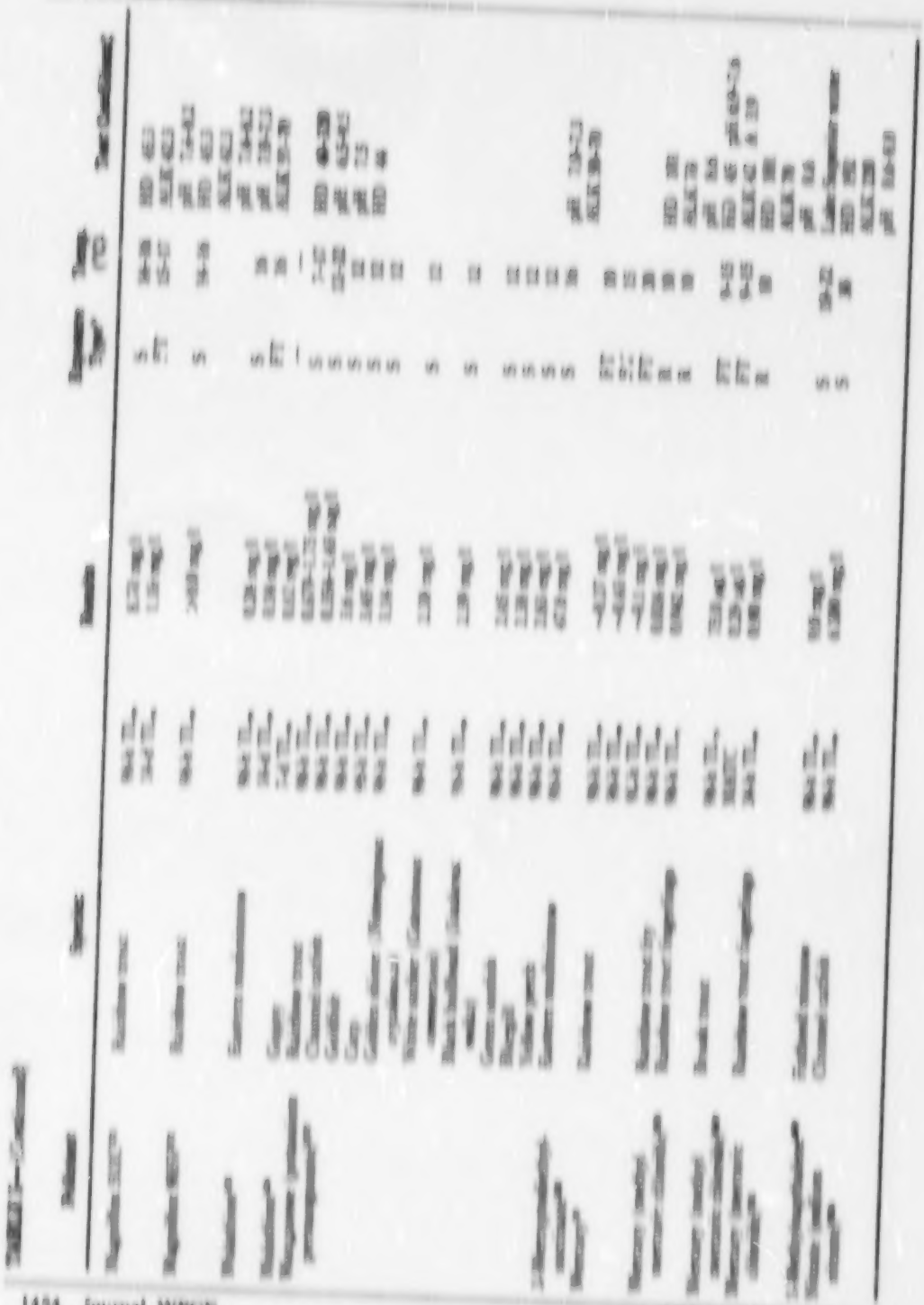
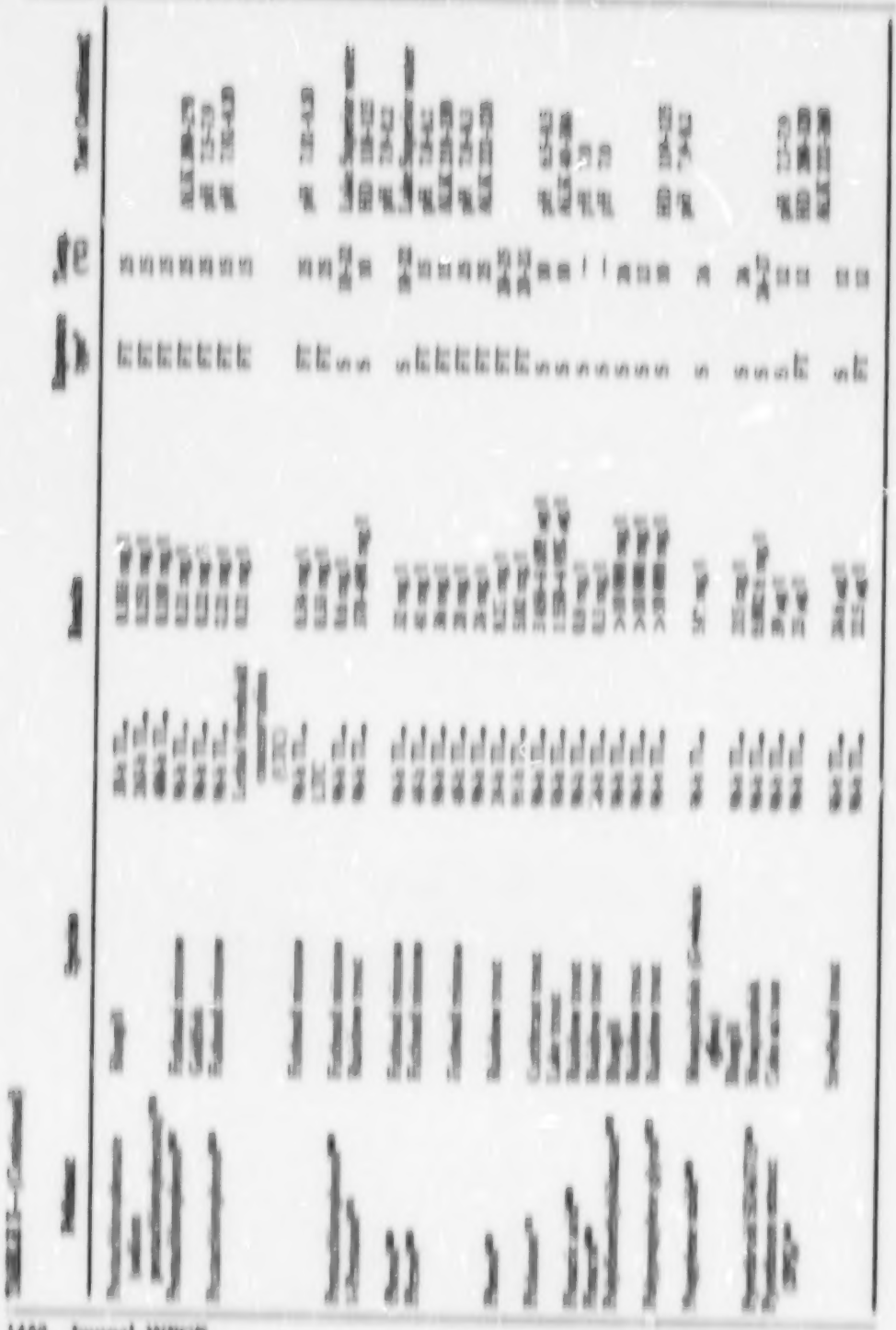
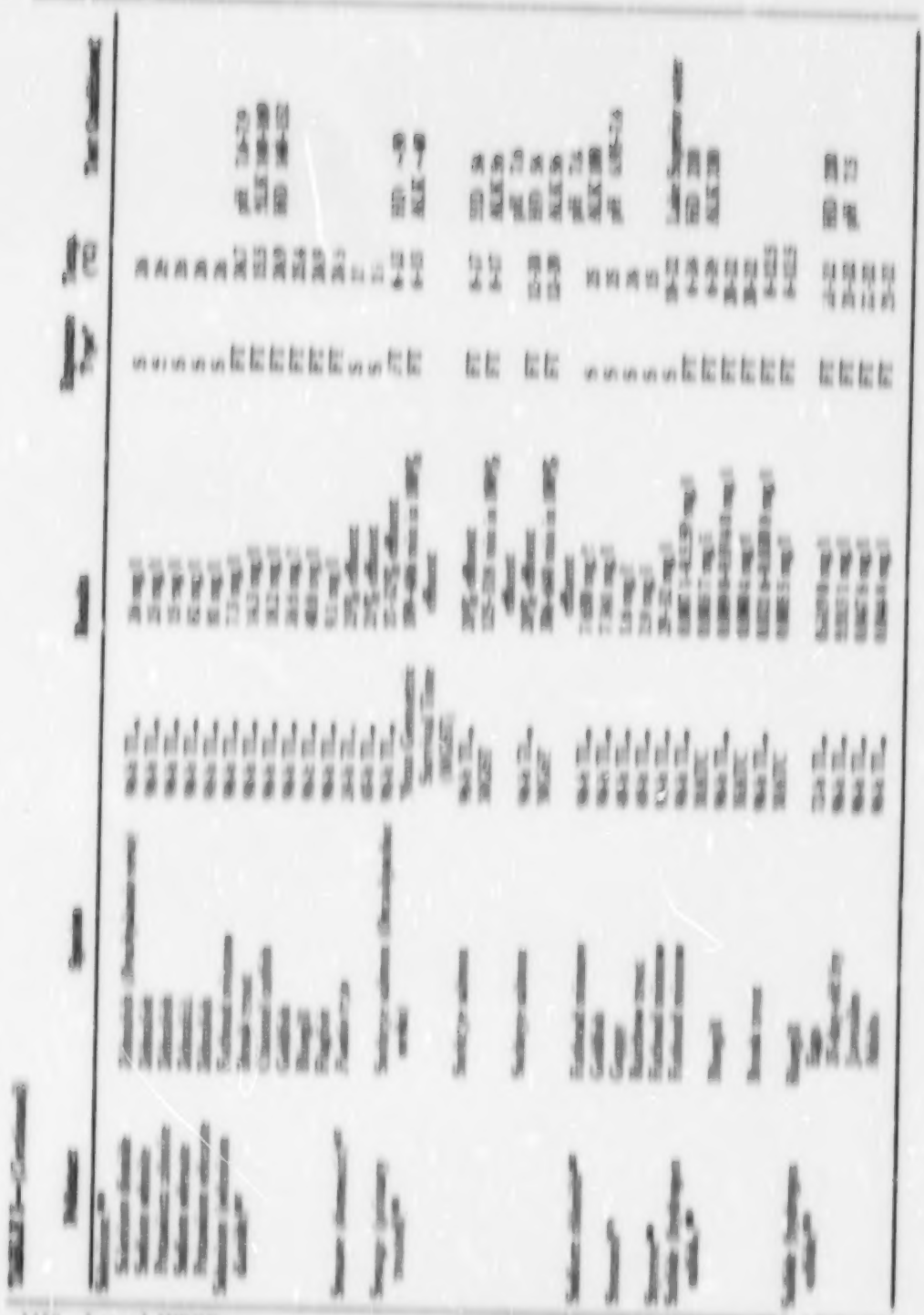


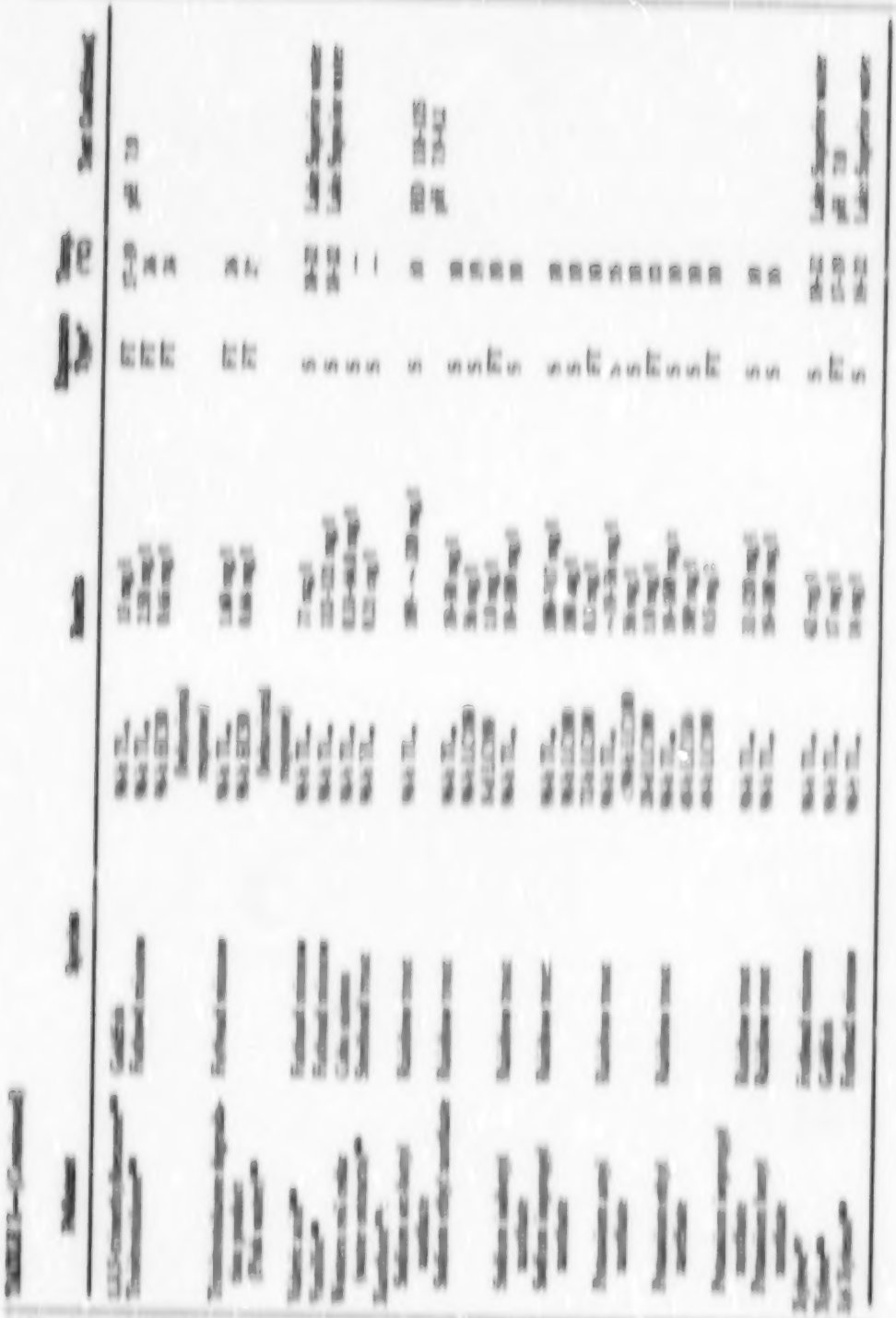
TABLE I—(Continued)

Pollutant	Species	Results		Exposure Type*	Temp. (°C)	Test Conditions†
Kraft mill bleachery effluent (6 mills)	Coho salmon	96-h TL50	79.0% effluent	FT	20-22	HD 5.0 ± 1.3
		96-h TL50	41.0% effluent	FT	20-22	ALK 3.6 ± 0.7
		96-h TL50	24.5% effluent	FT	20-22	pH 6.5 ± 0.2
		96-h TL50	12.5% effluent	FT	20-22	
Lead (lead nitrate)	Rainbow trout	96-h TL50	471-542 mg/l	S	14.5	HD 353 ALK 245
		96-h TL50	1.27 mg/l	FT	11.1	HD 27.7 ALK 29
		MATC	120 µg/l	FT	14.5	
		MATC	4.1 µg/l	FT	11.1	Test initiated with eyed eggs
		MATC	7.2 µg/l	FT	11.1	Test initiated after hatching and swim up of fry
Lead (lead nitrate)	Brook trout	96-h TL50	4,100 µg/l	FT	9-15	HD 44 pH 6.8-7.6
		MATC	58 µg/l	FT	9-15	ALK 43
Lindane	Rainbow trout	24-h TL50	0.051 mg/l	FT	20-25	pH 6.5 ± 0.2
		96-h TL50	0.032 mg/l	FT	20-25	ALK 3.8 ± 0.7 HD 5.0 ± 1.5
Lindane	Zebra fish?	96-h TL50	0.94? mg/l	S	20	pH 7.3
		96-h TL50	0.41? mg/l	S	22?	ALK 117?
		12-h TL50	0.87? mg/l	S	20?	
		12-h TL50	0.60? mg/l	S	22?	
Lindane	Bluegill	1-y TL50	20? µg/l	FT	27?	HD 37 pH 6.9
		MATC	9.1 µg/l	FT	27?	ALK 31 [illegible] 6.6
	Fathead minnow	1-y TL50	69 µg/l	FT	25	HD 46 pH 6.9
		MATC	9.1 µg/l	FT	25	ALK 35 [illegible] 6.7
	Brook trout	1.5-y TL50	26? µg/l	FT	9-16	HD 37 pH 6.9
		MATC	8.8? µg/l	FT	9-16	ALK 32 [illegible] 6.8
Linear alkyl benzene sulfonate	Carp	96-h TL50	5.6? mg/l (commercial grade + enzyme)	S	20	
		96-h TL50	5.0? mg/l (technical grade)	S	20	
	Tilapia	96-h TL50	4.6? mg/l (commercial grade)	S	20	
		96-h TL50	12.0? mg/l (commercial grade + enzyme)	S	20	
		96-h TL50	22.0? mg/l (technical grade)	S	20	









varied from 0.068 7 mg/l for rainbow trout to 0.084 mg/l for goldfish. Except for goldfish, the fry stage was up to 3 times more sensitive than the juvenile stage. Within the pH range of 7.1 to 8.7, 96-hour TL_m values decreased linearly with increased pH; however, log TL_m values increased linearly with increased pH in a pH range of 6.5 to 8.7. In chronic tests, maximum no-effect concentrations ranged from 0.000 4 mg/l for bluegills to 0.01 mg/l for goldfish. Sano [95] described the role of pH on the acute toxicity of sulfite to the guppy. The results showed that the toxicity of sulfite decreased with increased pH. Comparisons with data on goldfish showed that the dependence of sulfite toxicity on pH was weaker with the guppy than with goldfish. Robinette [97] evaluated the effect of sublethal levels of ammonia on channel catfish. Growth of fish was significantly reduced from the controls at 0.12 mg NH_3-N/l. The 24-hour T_{1m} value for catfish was 2.30 mg NH_3-N/l. The acute toxicity of nitrogenous compounds to channel catfish was evaluated by Colt and Tchobanoglous.[98] The results showed that the toxicity of un-ionized ammonia decreased at higher temperatures, while the toxicity of nitrous acid increased. The 96-hour TL_m values at 30°C for un-ionized ammonia, nitrite and nitrate were 1.9, 44, and 6 200 mg/l, respectively. Jones [99] determined the effects of various toxicants on guppies and described methodology for toxicity tests. Concentrations, period of exposure, and reactions were recorded. The toxicants included several inorganics, organics, and pesticides. The use of fathead minnows and goldfish as standard fish in bioassays and the reaction to potential reference toxicants was evaluated by Adelman and Smith.[107] Fathead minnows were recommended on the basis of small size and capability for use in complete life cycle tests. On the basis of minimum variability of bioassay results, NaCl was superior for use as a reference toxicant. Both NaCl and pentachlorophenol seemed capable of detecting abnormal fish.

The effects of selected water toxicants on the in vitro activity of channel catfish carbonic anhydrase were studied by Christensen and Tucker.[100] In general, enzyme activity was strongly reduced by heavy metal inorganic cations, less strongly by organometallic cations, some anions, and certain pesticides, and weakly inhibited by light metal cations, organometallic, and organic compounds. Matsumura *et al.*[101] investigated the incorporation of ^{203}Hg into methylmercury in fish livers from salt water and freshwater fishes (northern pike and

chinook salmon). It was found that livers from all fish, particularly pelagic species, were capable of transforming mercuric ion into methylmercury *in vitro*. The factors involved in the process of methylmercury formation were studied. Wobeser [15] examined the effect of prolonged oral administration of methylmercuric chloride to rainbow trout fingerlings. Fish exposed to 18 and 34 mg Hg/l as methylmercuric chloride over a 105-day period had significantly higher blood packed cell volumes than did controls. Hyperplasia of the gill epithelium was the only morphologic alteration detected in these fish. Individual fish accumulated up to 30 μg Hg/g in muscle tissue, but no mortality attributed to Hg occurred. Brown [16] investigated the effects of some pollutants on enzymes of aquatic organisms. Data on rainbow trout liver ornithine aminotransferase activity showed that activity was inhibited proportionate to increased Hg concentration. A short review was provided on the adaptation of fish to some inorganic and organic pollutants. The binding of Hg and Zn to Cd-binding protein (Cd-BP) in the liver and kidney of goldfish was investigated by Marafante.[17] Hg and Zn besides Cd, were incorporated into Cd-BP I and in the liver than in the kidney (1.8 percent), while Hg showed similar affinity for the Cd-BP of these organs (17 and 18 percent, respectively). The acute effects of Hg and Cu on the olfactory responses of rainbow trout were determined by Hara *et al.*[18] Olfactory responses were depressed within 2 hours during exposure to 0.10 mg Hg/l and 0.008 mg Cu/l. The depression increased with increased concentration and exposure time. Recovery of this response was slower with the higher concentrations and longer exposure.

The effects of Cu or Zn on premigrant yearling coho salmon adaptability to seawater, ATPase activity, and effects of Cu on downstream migration were examined by Lorz and McPherson.[19] Exposures of coho salmon for 144 hours to Zn in fresh water had little effect on Na-K activated ATPase activity in gill microsomes or on the survival of fish transferred to sea water. Acute and chronic exposures of Cu (5 to 50 μg/l) in fresh water had deleterious effects on downstream migration, gill ATPase activity, and survival in sea water. Pasquinot [20] determined the effect of Cu on mucus cells in alevin trout (*Salmo trutta*). A decrease in the number of stained mucus cells in the skin and intestine was noticed from fish exposed to 0.1 mg/l for 10 days. The mechanism of action of Cu upon juvenile striped bass (*Morone saxatilis*) was defined by Courtois.[21] Ambient exposure of striped bass to Cu caused a breakdown in normal osmotic homeostasis. The breakdown in water balance of the Na-H exchange sites on the gill was thought to be the mode and site of action of Cu. The effect of Cu on the appetite, growth, and proximate body composition of rainbow trout held in hard water (308 mg/l) for 40 days was studied by Lett *et al.*[22] Food intake and growth rate were initially depressed at concentrations of 0.1 to 0.3 and 0.073 to 0.535 mg Cu/l, respectively, but approached values observed for control fish near the end of the 40-day period. Initial growth retardation was not attributable to the inability of exposed fish to digest their daily rations. Lipid, protein, and moisture of fish exposed to Cu did not change significantly during this period. Mukherjee and Bhattacharya [23] investigated the effects of industrial pollutants on the hepatopancreas of two common Indian freshwater fish (*Ophiocephalus punctatus* and *Clarias batrachus*). Results showed that 48-hour TL_m concentrations of all pollutants caused severe histopathological lesions with Cu causing the most deleterious effects. More severe pathologic conditions were observed after 90 days of exposure. McCarty and Houston [24] reported the effects of Cd upon the water electrolyte status (plasma and muscle levels of Na, K, and Cl, and muscle water content) in goldfish. Significant changes in plasma chloride, tissue K, and tissue water content were observed after 35 days to 44.8 and 380 μg Cd/l. Goldfish exposed to 380 μg/l for 50 days exhibited significant deviations in plasma Na and Cl levels, as well as in tissue Na and water content. These parameters were thought to provide useful indices of Cd effects at sublethal levels. Dose response studies to relate specific quantities of Cd to histopathological effects in goldfish were conducted by Tafaroli and Summerfelt.[25] The 96-hour TL_m for intraperitoneal injections of Cd was 30.0 μg/l wet weight. Examination of gonadal tissue receiving multiple injections of Cd indicated histopathological changes, including damage to germ cells and seminiferous tubules. It was concluded that Cd may have serious effects on immunoregulation, hematopoiesis, and gametogenesis. The histological and biochemical responses of the mummichog (*Fundulus heteroclitus*) to combinations of levels of Cd and O_2 in fresh water were reported by Vyas *et al.*[26] Levels of mortality were related to duration of exposure, Cd concentration, and levels of O_2. Median tolerance concentrations,

at 96 hours ranged from 1.3 to 3.0 mg Cd/l at 2.3 and 8.8 mg ca/l, respectively. Histopathology was evident in fish exposed to 25 mg Cd/l for 0.5 hours, but not in fish exposed to 3 mg Cd/l. Comparisons of the results of this test with those of others conducted in sea water show that mummichogs were more resistant to Cd in sea water than in fresh water. Sarot and Perlmutter[153] determined the effect of Zn on the immune response of sockeye to infectious pancreatic necrosis virus (IPN) and to *Proteus vulgaris*. Zinc appeared to suppress the immune response against *Proteus vulgaris*, but not against IPN. The effects of Zn on Δ^5-3β-hydroxysteroid dehydrogenase (Δ^5-3βHSDH) activity in the head kidney of rainbow trout were studied by Watson and McKeown.[154] Trout exposed to 0.048 mg/l showed increased Δ^5-3βHSDH activity compared to controls. The increase in activity was associated with the stimulation of the pituitary-interrenal axis by the noxious stress of Zn. Temperature effects on lactate-glycogen metabolism in Zn-intoxicated rainbow trout was assessed by Hodson.[155] Lactic acid production and glycogen utilization in dorsal white muscle of trout increased with exposure time to a lethal concentration (14 to 16 mg/l) of Zn. Exposures at 5°, 11°, and 19°C did not affect the timing or percent change of lactic acid production or utilization of glycogen. The previously known high tolerance to Zn by warm-acclimated fish was not caused by increased resistance to tissue hypoxia caused by gill damage. The activity of δ-amino levulinic acid dehydratase in blood of Pb-exposed rainbow trout was measured by Hodson.[156] Pb concentrations of 13 μg/l caused a significant inhibition of activity after a 4-week exposure. It was concluded that this enzyme's activity may provide a short-term indication of long-term harmful effects of Pb. Structural alterations in rainbow trout epidermal mucus produced by water-borne Pb and Hg were studied by Varanasi *et al.*[157] Results indicated that Pb and Hg produced significant alterations (fluidization) of epidermal mucus of these fish. Large differences were shown to exist in the degrees of accumulation and depuration of both metals with structural alterations remaining after a period of depuration. Forensen[158] observed the ultrastructural changes in the hepatocytes of green sunfish when exposed to solutions of sodium arsenate. The level to which As was concentrated was positively associated with water concentration, duration of exposure, and water temperature, and as As concentration and exposure time increased, the numbers of intranuclear and cytoplasmic electron dense particles and aberrant mitochondria increased. In addition, lysosomes and smooth endoplasmic reticulum increased in number, while the number of myelin figures decreased. Penrose[159] examined the biosynthesis of organic As compounds in brown trout. Radioactive, inorganic As was administered to trout orally and by intramuscular injection. Orally administered As appeared in the tissues in a presumed organic form which was also found in the gastrointestinal contents. Injected As appeared in the tissues in inorganic form and was slowly converted to organic form; both inorganic and organic As were detected in the bile. Conversion to "organic" form was mediated by the intestinal flora. The effect of $Fe(OH)_3$ suspension on blood chemistry in the common shiner (*Notropis cornutus*) was investigated by Brenner *et al.*[160] Concentrations of 3 mg $Fe(OH)_3$/l for periods of 2 to 8 weeks resulted in initial changes in serum protein, glucose, Na, and K, but these changes did not cause mortality or gill damage. The metabolism of Fe by normal and Fe-deficient rainbow trout was investigated by Walker and Fromm.[161] In both groups, most of the ^{59}Fe was absorbed from the peritoneal cavity within 24 hours, and equilibrium between plasma ^{59}Fe pool and that in the tissues was obtained 5 days after an intraperitoneal injection of 1.0 μCi ^{59}Fe/100 g fish. In Fe-deficient fish, the ^{59}Fe content of RBC increased to 70 to 80 percent of the injected dose by Day 10, compared to 50 percent in controls. An average of 15 percent of injected ^{59}Fe was taken up by hepatic tissue of control fish but was reduced to 1 percent of the initial dose by Day 10 post bleeding in Fe-deficient trout liver. Smissaert *et al.*[162] provided a brief overview of some of the multitissue injury problems met in an experiment on the effect of trivalent Cr on rainbow trout. Acetylcholinesterase (AChE) activity in young trout exposed to 10 mg Cr/l for 30 days appeared to be decreased from the control, but difficulties in the interpretation of the results were discussed. Histopathological examination of the gill did not reveal any abnormalities. An important finding was that AChE activity was extremely high, even when compared with nervous tissue. Stelk *et al.*[163] observed the toxicity of hexavalent Cr to the roach and rainbow trout with special reference to organ weights, liver and plasma enzyme activities, blood parameters and histological alterations. Fish exposed to 10 mg Cr/l for 3 to 4 weeks showed increased Na, glucose, hematocrit, and hemoglobin content of the blood. Gill epi-

thelium hypertrophy and hyperplasia were also shown. In addition, this concentration caused impaired gill and kidney function, along with intestinal hemorrhages and lysis of the intestinal epithelium in both species. The effect of in vitro Cr exposure on Na-K- and Mg-ATPase activity in tissues of rainbow trout was studied by Kuhnert *et al*.[113] The results indicated that in vitro exposure to hexavalent Cr inhibited Na-K-ATPase activity in several trout tissues. The inhibition of this enzyme may partially explain the toxicity of hexavalent Cr to fish. Smith and Russo[114] exposed rainbow trout to concentrations of $NaNO_2$ to determine the minimum concentration that induces methemoglobinemia (M-Hb) in the blood. When compared with controls, exposure to 0.080 mg NO_2/l and above for 8 days resulted in significant elevation of M-Hb. Significant differences in total Hb concentrations were not observed. The effect of thiocyanate (SCN) on Cl and Na fluxes across the gill of goldfish was studied by Rankin.[115] At low external Cl concentrations, the addition of SCN markedly inhibited Cl influx and efflux, reversing the net flux. Thiocyanate had no effect on Na fluxes when injected into the urinary papilla of fish or added to the external medium. A kinetic model was proposed to explain Cl and HCO_3 transport across the gill of these fish. Schulze-Wichenbrauk[116] investigated the effects of sublethal un-ionized ammonia concentrations on metabolism in juvenile rainbow trout. Concentrations up to 0.13 mg NH_3/l in hard fresh water did not adversely affect growth, food consumption, and food conversion within 3 to 7 weeks. Fish were temporarily affected at concentrations greater than 0.15 mg/l. Acclimation to sublethal ammonia concentrations raised the resistance of trout lethal ammonia concentrations, depending on size and feeding condition of the fish. The effect of un-ionized ammonia on the gill structure of rainbow trout was studied by Smart.[117] Gill damage resulting from acute exposure to 0.50 to 0.65 NH_3/l was relatively minor and was not thought to be the primary cause of death. Severe histopathological changes in gill structure were observed, however, when fish were exposed to 0.25–0.30 mg NH_3/l for longer than 14 days. Changes in gill structure in these exposures were thought to be a principal cause of death because of impaired O_2 uptake. High incidences of disease were thought to be an indirect long-term effect.

Behavioral responses of whitefish (*Coregonus clupeaformis*) and rainbow trout to drilling fluids containing several inorganic compounds were examined by Lawrence and Scherer.[118] Some tests were run with infra-red light to separate photic effects from chemical effects. Both whitefish and trout showed preference for mud suspensions at the maximum concentration tested (1 000 g/l), and both species showed a shift in reaction to supernatant from preference at 1 000 g/l to avoidance at 10 000 g/l. The avoidance of lime-neutralized iron hydroxide solutions by coho salmon was studied by Updegraff and Sykora.[119] Definite avoidance reaction of these fish to suspensions of 4.25 to 8.40 mg Fe/l was noted. Fish exposed for several months to different concentrations of the pollutant showed an almost identical response as fish raised in the controls. The rate of O_2 consumption by sticklebacks (*Gasterosteus aculeatus*) exposed to Zn was studied by Brafield and Matthiessen.[120] Exposure to 1 mg Zn/l in Ca-free water caused wide variations in individual responses, but O_2 uptake tended to rise and then become extremely erratic before declining as death approached. Behavioral abnormalities, such as increased ventilation rate, loss of balance, and long periods of inactivity alternating with spasmodic swimming, also occurred. If restored to Zn-free water after 40 hours exposure to Zn, recovery was generally complete, although fluctuating rates of O_2 uptake persisted.

Organics. Isleib[121] summarized Great Lakes Environmental Contaminants Survey data for the concentrations of PCBs, DDT, dieldrin, and Hg in fish for 1972 and 1973. Concentrations of PCBs were generally twice those for DDT in 1972. Catfish, chub, coho salmon, lake trout, and walleyes from one or more locations exceeded FDA action guidelines for one or more substances. In 1973, catfish, carp, lake trout, walleye, and white bass exceeded FDA action guidelines. The major problem in Lake Michigan was PCBs. Bails[122] discussed the role and the problems of the Great Lakes Environmental Contaminants Survey in sampling and analyzing fish from the Great Lakes for DDT and metabolites, PCBs, dieldrin, and Hg. In a survey of suburban Lake Anne watershed, Reston, Va., Martell *et al*.[123] reported PCB residue concentrations as high as 0.550 g/g and 0.700 g/g in bluegills (*Lepomis macrochirus*) and largemouth bass (*Micropterus salmoides*), respectively. Allright *et al*.[124] found, in decreasing order of magnitude, PCBs, p,p'-DDE, heptachlor epoxide, o,p'-DDT, and p,p'-DDD in fish, crabs, and shellfish collected from the lower Fraser River, its estuary, and selected areas of Georgia Strait in British Columbia. The great-

est concentrations occurred in biota from the waters adjacent to Vancouver. Animals from Georgia Strait and from areas away from the immediate influence of the Fraser River contained no detectable levels of chlorinated hydrocarbons. Johnston *et al.*[107] also reported on the concentrations of chlorinated hydrocarbon pesticides and PCBs in fishes from the lower Fraser River. The mean PCB concentration in the epaxial white muscle tissue of 183 specimens of 13 species of fish was 0.14 μg/g, the highest residue level for any chlorinated hydrocarbon measured. Kveksata[108] measured levels of Aroclor 1254 (as high as 413 μg/l) in the rivers Avon and Frome in the Bristol area, United Kingdom, in 1973. Aroclors 1242, 1254, and 1260 were found in fish from the River Avon. Concentrations of Aroclors 1254 and 1260 in livers and visceral fat of bleak and pike ranged from 10.8 to 20.4 μg/g. A study of the environmental distribution of PCBs in the Ashida River, Japan, was conducted by Nakagawa *et al.*[67] Residues of PCBs in fish and shellfish were 0.01 and 0.24 μg/g, respectively. Norstrom *et al.*[110] developed a pollutant accumulation model based on the pollutant's kinetics and coupled to fish energetics which successfully predicted the concentration of PCBs and methylmercury in tissues of yellow perch (*Perca flavescens*) from the Ottawa River, Canada. Zitko[111] described the contamination of the environment with organochlorine compounds and discussed the modes of transport in the environment, availability, particularly when adsorbed on ss, metabolism and possible metabolites, and the toxicological information which needs to be generated for these compounds in relation to fish and the aquatic environment.

Fish, milk, egg, and vegetable samples were collected by Yip[112] within a 42-km radius of 10 perchloroethylene or trichloroethylene manufacturing plants and analysed for hexachloro-1,3-butadiene (HCBD). Concentrations of 0.01 to 1.3 μg HCBD/g were found in nine fish caught in the lower Mississippi River. Laska *et al.*[113] in another study on the lower Mississippi, reported residue concentrations of 0.072 to 0.38 μg HCBD/g in mosquitofish (*Gambusia affinis*) collected during a survey of the distribution of hexachlorobenzene and HCBD in water, soil, and selected aquatic organisms. Oehlbach *et al.*[114] identified HCBD residues in fish from inland water fed by the Rhine, particularly in the Ketelmeer. Residue levels were related to fat content, but no biomagnification in the food chain was observed. Concentration in the fish of HCBD ranged from 0.11 to 5.04 μg/g. The level of HCBD in the water of the Ketelmeer was 0.15 μg/l. Yurawecz *et al.*[115] modified a method for pesticides for the determination of HCBD in fish.

Octachlorostyrene was isolated and identified by Kuehl *et al.*[116] in walleye (*Stizostedion vitreum vitreum*) from Lake Huron and the lower Detroit River, and from alewife (*Alosa pseudoharengus*) from Lake Ontario. Schultz and Tebo[117] described a biological survey conducted on the effects of a 28-kiloliter diesel fuel spill into Boone Creek, S. C. An estimated 90 percent of the fish population was killed during the initial spill. Analysis of substrate sediment samples revealed hydrocarbons still present 13 months after the spill.

A method for the determination of PCBs, terphenyls, and naphthalenes using silicic acid chromatography, chlorination, and gas chromatography (GC) was described by Vanucchi *et al.*[118] Zitko[119] described a rapid screening method for detecting the presence of polynuclear aromatic hydrocarbons in aquatic fauna. The detection limit for crude, Bunker C, and creosote oil with the method was 100, 50, and 100 μg lipid/g, respectively. Statham *et al.*[120] described a technique for monitoring certain organic pollutants in aquatic environments by analysis of fish liver bile and demonstrated the usefulness of the technique. Bile to water radioactivity ratios as high as 10 000:1 were found after 24-hour exposures of rainbow trout (*Salmo gairdneri*) to nine different radioactive organic compounds. Solvent extraction, steam distillation, and capillary column gas chromatography-mass spectroscopy (GC-MS) was used by Connell *et al.*[121] to identify hydrocarbons in fish fillets.

Metcalf *et al.*[122] studied the degradation and biomagnification of radiolabeled tri-, tetra-, and pentachlorobiphenyls, and DDE by algae, snails, mosquitos, and mosquitofish for 33 days in a laboratory model ecosystem. Trichlorobiphenyl was most rapidly degraded, while pentachlorobiphenyl was about as persistent as DDE. A linear relationship was found between lipid:water partition and between water solubility and ecological magnification. Mosquitofish showed an ecological magnification factor of 8 400, 11 583, 12 152, and 12 037 for tri-, tetra-, and pentachlorobiphenyl, and DDE, respectively. The accumulation and metabolism of 2,2-dichlorobiphenyl-^{14}C in goldfish (*Carassius auratus*) in a simulated ecosystem were studied by Hutzler *et al.*[123] The fish were capable of only limited metabolism compared to the other organisms in the test system. Santhon *et al.*[124] reported

on the fate of di-n-octyl phthalate (DOP) in a 35-day terrestrial-aquatic and 3-day aquatic model ecosystem, and on *in vitro* metabolism studies of DOP in liver homogenates of mosquitofish. Ecological magnification factors were 0.400 and 7.19 for the mosquitofish in the 35-day and 3-day model ecosystems, respectively. The increased accumulation value in the 35-day system indicated the persistence of the material and its long-term unfavorable accumulation by aquatic organisms. The preferred metabolic pathway of DOP determined by the *in vitro* studies of liver homogenates was non-oxidative ester cleavage.

Branson *et al*.[35] developed and evaluated an accelerated test procedure, based on kinetics, for measuring the bioaccumulation potential of chemicals by fish. Comparisons of steady state bioconcentration factors for 2,2',4,4'-tetrachlorobiphenyl, using the accelerated test procedure and a 42-day test of rainbow trout in a flowing water system, showed good agreement. The measured 82 ± 20 $\mu g/g$ concentration of 2,2',4,4'-tetrachlorobiphenyl in trout muscle for the 42-day exposure to 14 $\mu g/l$ was within the experimental error of 92 ± 18 $\mu g/g$ calculated from the accelerated test data. White sucker (*Catostomus commersoni*) accumulated Aroclor 1232 and dieldrin at similar rates, according to Frederick,[36] but at similar concentrations accumulated PCB levels were higher. Canton *et al*.[37] studied the short- and long-term toxicity and the accumulation and elimination of α-hexachlorocyclohexane for separate organisms (algae, crustacean, and fish) from different trophic levels and for the food chain. For *Daphnia magna*, guppy (*Poecilia reticulata*), and rainbow trout the 48-hour TL_m values were 800, 800 and 1 050 $\mu g/l$, respectively. Compared with contamination by water alone, the transference from *Daphnia* to guppy was 3 to 4 times higher, 420- to 1 000×, compared to 140 to 250× for concentrations of 10 to 80 $\mu g/l$ in water. Rainbow trout showed no toxicity for up to 1 250 $\mu g/g$ in their diet for 3 months based on mortality, growth, enzyme activities, or histopathology. Ogata and Miyake[38] investigated the accumulation of benzene, toluene, m- or p-xylene, and o-xylene in eels (*Anguilla anguilla*) after exposure to Arabian light crude oil in a static renewal test system. When expressed as the ratio of the compound in eel flesh to the compound in the exposure water, toluene was accumulated in the highest concentrations, followed by o-xylene, m- or p-xylene, and benzene in decreasing order. Eels exposed to oil could be distinguished from controls in taste tests. Khalil *et al*.[39] studied the absorption of a series of barbituric acid derivatives using overturn times of the mosquitofish in static tests, and observed a correlation between absorption and solubility of the derivatives. Effects of pH and temperature were also studied for some of the derivatives.

In a series of papers, the absorption, accumulation, and excretion of phenol by goldfish were reported by Kobayashi and Akiyake[40] and Kobayashi *et al*.[41, 42] No mortality occurred at 5 to 80 mg phenol/l, but 10 percent mortality occurred at concentrations of 40 to 100 mg/l. Absorption and excretion curves were similar for 20 mg phenol/l, with most of the absorption and excretion occurring within the first hour. Concentration factors for the 3-day test ranged from 1.5× to 3.3× for live fish at 20 mg/l. Dead fish exposed to concentrations from 40 to 100 mg phenol/l had phenol concentrations in the tissues of 68 to 187 $\mu g/g$. Although phenol was initially excreted in the free form, the ratio of conjugated to free phenol increased with time. Phenol concentrations in all tissue except the gall bladder were almost constant after a 9-hour exposure to 15 mg phenol/l, and decreased rapidly in clean water. The large accumulation and slow decline of phenol to the gall bladder, however, indicated biliary excretion and probable detoxification by sulfate conjugation. A conjugated phenol, p-enylsulfate, excreted from goldfish exposed for 14 hours to a medium containing 15 mg phenol/l was isolated and identified. Contrary to reports of glucuronide conjugation of some phenolic compounds in fish, no glucuronides were detected during this study.

Hansen *et al*.[43] evaluated the toxic effects and the selective accumulation of PCB components by fingerling channel catfish (*Ictalurus punctatus*) fed a diet containing 20 μg Aroclor 1 242/g for 250 days. Treated fish showed reduced weight gain and hypertrophy of the liver during the first 20 weeks. After treatment was discontinued temporarily for 8 weeks, weight gain surpassed and liver weight approached the control results. Polychlorinated biphenyls shifted from edible carcass to offal during the PCB-free period. Histological examination of tissues failed to disclose tissue alterations. Juvenile coho salmon (*Oncorhynchus kisutch*) were fed 2,5,2',5'-tetrachlorobiphenyl, 2,4,5,2',4',5'-pentachlorobiphenyl, and 2,4,5,2',4',5'-hexachlorobiphenyl by Groger *et al*.[44] at a combined total concentration of 1, 5, and 15 $\mu g/g$ for 71 days. Salmon fed 15 $\mu g/g$ for 30 days gained significantly more

weight than controls. No such difference was observed at the other treatment concentrations. Tissue concentrations of salmon fed 1 μg chlorobiphenyls/g reached apparent equilibrium conditions. In a separate experiment, pentachlorobiphenyl enhanced activity of hepatic aryl hydrocarbon hydroxylase in treated fish. Fingerling rainbow trout were fed a diet fortified with 10 μg Chlorowax 500(C)/g for 82 days by Lombardo *et al.*[111] No gross toxicological effects were noted, although the experimental fish gained significantly less weight than controls. Chlorinated paraffin residues as high as 1.1 μg/g were found in the experimental fish.

Hogan and Brauhn[112] found 0.7 μg/g of Aroclor 1242 and 0.09 μg/g of the DDT complex in eyed rainbow trout eggs from a hatchery brood stock. Studies showed 60 to 70 percent of the fry were deformed 90 days after hatching, and cumulative mortality was 75 percent 30 days after receipt of the eggs. Poels and Strik[113] exposed rainbow trout to Rhine River water for 9 months. Increased somatic liver and kidney index, increased blood glucose content, and stenosis occurred. Strong accumulation of PCBs, HCB, and pentachlorobenzene occurred in adipose tissue after 6 months exposure, with concentrations ranging from 1.7 to 10 μg/g. Mann[114] described an acute toxicity test with the golden orfe suitable as a routine technique for assessment of toxicity of substances in water and effluent. The results of round-robin tests by seven laboratories of 18 chemicals from heterogeneous groups of substances were presented.

Leeuwangh *et al.*[115] found 96-hour TI_m values of 90, 130, and 210 μg HCBD/l for goldfish, an aquatic sowbug (*Asellus aquaticus*), and a snail (*Lymnaea stagnalis*). In a 49- to 67-day sublethal exposure with goldfish, decreased body weight at 30 μg/l, increased relative liver weight, and changes in liver enzyme activities at 0.6 μg/l occurred, but no significant changes were detected at 0 μg/l.

Slipp[116] found TI_m values 2.6 times higher when phenol was slowly added to a test system containing fingerling channel catfish than when the fish were directly immersed in the test solution. No effect on fathead minnow (*Pimephales promelas*) reproduction occurred after single 24- and 48-hour exposures to phenol, while 10 and 20 mg phenol/l caused significant increases in breathing rates of bluegills. Adelman *et al.*[117] determined the difference in sensitivity to pentachlorophenol of different-sized fathead minnows and goldfish of a constant age or stock, and the sensitivity of different-aged fathead minnows to flow-through tests. Differences in threshold TI_m values for large and small goldfish were small, but significant. Threshold TI_m values were not significantly different for fathead minnows of different size or age. Static 96-hour laboratory bioassays were conducted by Mattson *et al.*[118] with 50 organic compounds commonly used by industry. The selected compounds represented the five following chemical classes: acids, alcohols, hydrocarbons, ketones, aldehydes, and phenols. The most lethal chemical to the fathead minnows was pentachlorophenol (96-hour TI_m of 0.6 mg/l) and the least lethal was ethanol (96-hour TI_m of 13 400 mg/l).

Silva[119] studied the acute toxicity of dioctyl phthalate to rainbow trout and found 100 percent survival for 48 hours in static tests at concentrations as high as 1 000 mg/l. The acute toxicity of propylene oxide to mosquitofish and bluegills, and a marine species, was evaluated in static tests by Crews.[120] The 96-hour TI_m values were 141 and 215 mg propylene oxide/l for the mosquitofish and bluegill, respectively. Smock *et al.*[121] found alpha trinitrotoluene (alpha TNT) and its degradation product pink water (TNTco) were 2 to 25 times more toxic to fathead minnows than to algae. The 96-hour TI_m values (for flow-through tests) with fathead minnows were 2.58 and 1.60 mg/l for TNT and TNTco, respectively. In addition to egg mortality, gross anatomical malformations were found by Hyman *et al.*[122] in 1.9, 2.1, 2.7, and 4.3 percent of newly hatched carp (*Cyprinus carpio*) exposed from fertilization to 0.5, 1.0, 5.0, and 10.1 mg 5-chlorouracil/l in static tests. In analyses of chlorine-disinfected waste, 5-chlorouracil was found as a by-product.

Brenniman *et al.*[123] used a continuous flow system to evaluate the effects of outboard motor exhaust and selected volatile aromatic toxicants on goldfish. TI_m values (96-hour) of 171, 168, 23, 17, and 13 mg/l were calculated for leaded outboard motor exhaust, non-leaded exhaust, toluene, xylene, and 1,3,5-tri-methylbenzene, respectively. Yearling rainbow trout were exposed by Lockhart *et al.*[124] to saturated solutions of tri-aryl phosphate lubricating oil in a long-term continuous exposure. Guppies were also exposed in a 24-hour static test. No mortality occurred in the guppy test, but the yearling trout in the long-term test refused floating food after 14 days, and showed a 3- and 6-fold increase in serum lactate dehydrogenase and glutamic oxaloacetic transaminase activity and reduced fatty tissue after a 4-month exposure. Arshantlas[125] re-

ported on the toxicity of treated petroleum processing wastes to eggs, larvae and adults of *Salmo trutta*, brown trout (*Salmo trutta*), and the carp. The toxic effects included mortality and inhibited growth or development for all stages. Rice *et al*[106] determined the acute toxicity of Prudhoe Bay crude oil on eggs, alevins, and fry of pink salmon (*Oncorhynchus gorbuscha*). Sensitivity to oil increased during development. The 96-hour TL_m values for eggs, mid-alevins and emergent fry were 0.82, 0.55, and 0.40 ml oil/l. Growth in sublethal tests varied with exposure concentrations and stage of alevin development. The lowest concentration tested, 0.078 ml oil/l, caused reduced fry growth.

Lopez-Zavala *et al*[107] evaluated the acute toxicity of technical grade and commercial grade formulations of alkyl benzene sulfonate (ABS), linear alkyl benzene sulfonate (LAS), and alpha olefin sulfonate (AOS) to carp and *Tilapia melanopleura*, plants, and domestic pigs. In all the static tests with fish, technical and commercial grade formulations of ABS showed the lowest toxicity, while AOS formulations showed the highest toxicity for both carp and *Tilapia*. The 96-hour TL_m values for technical grade ABS, LAS, and AOS were 12.5 and 20.0 mg ABS/l, 5.4 and 23.0 mg LAS/l, and 4.0 and 8.0 mg AOS/l, for carp and *Tilapia*, respectively. The effect of detergents on rainbow trout and carp was studied by Rueger[108] and trout fry were found to be more sensitive than carp. A significant effect of DO on detergent toxicity was also demonstrated.

Biesinger *et al*[109] conducted static and flow-through acute toxicity tests with selected polyelectrolytes using rainbow trout, lake trout (*Salvelinus namaycush*), a mysid (*Mysis relicta*), and a copepod (*Limnocalanus macrurus*), and acute and chronic tests with a cladoceran (*Daphnia magna*). Superfloc 330 was found to be the most toxic to fish, with a 96-hour TL_m value of 2.0 mg/l for lake trout. Cyanamid-162 was the least toxic to fish, with a 96-hour TL_m of 216 mg/l for rainbow trout. The environmental impact of a selected group of polydimethylsiloxane compounds on several different biological systems was studied by Hobbs *et al*[110]. Static 96-hour TL_m values for both bluegill and rainbow trout were greater than 10,000 mg/l for Dow Corning Antifoam C Emulsion. Residue data from a 30-day renewal exposure with 1 and 10 mg/l Dow Corning 200 fluid 350 cs emulsion indicated no relation between exposure time or exposure concentration and residue levels. It was suggested that residues detected might have been caused by surface contamination. Welsh *et al*[111] in tests of mining flotation agents, found large differences in toxicity of eight collectors (xanthate) and four frothers to rainbow trout as measured by 96-hour static and 28-day flow-through exposures. Sodium ethyl and potassium amyl xanthate were the most toxic collectors, with 96-hour TL_m values ranging between 30 and 50 mg/l. Xylenol was the most toxic frother, with a TL_m between 3.0 and 3.2 mg/l. Long-term flow-through tests of some of the compounds indicated toxicity 100 times greater than in static tests.

Canton and Verzesch[112] determined the 48- or 96-hour toxicity of 18 feed additives to algae, *Daphnia magna*, guppies, and rainbow trout. Robenidine was the most toxic chemical to all organisms tested, and produced 48-hour TL_m values for *Daphnia*, guppies, and trout of 0.071, 0.8, and 0.078 mg/l, respectively. For pyrimethamine and sternurol, the next most toxic chemicals, 48-hour TL_m values were 7.5 and 5.0 mg/l and 1.0 and 3.9 mg/l for guppies and rainbow trout, respectively. Leach *et al*[113] found 96-hour TL_m values of 0.8, 1.8, 1.8, and 2.0 mg/l for Δ^9-dehydrojuvabione, juvabione, dihydrojuvabiones, and juvabiols, naturally-occurring insect juvenile hormone analogues, in static renewal tests with rainbow trout. All fish, including survivors, became lethargic and the skin darkened soon after exposure to the test compounds. Results of aminopyridine (a bird-frightening agent) exposures of birds and fish were reported by Schafer and Marking[114]. The 96-hour static TL_m values for both channel catfish and bluegill ranged from 2.43 to 7.96 mg aminopyridine/l at temperatures from 12° to 24°C and in very soft to hard water. Toxicity was significantly greater in very hard water and mortality more rapid at 24° than at 12°C.

Motohashima and Sugimura[115] reviewed the use of zebrafish (*Brachydanio rerio*) and guppy in experimental carcinogenesis studies of nitrosamines, aromatic amines, polycyclic hydrocarbons, mycotoxins, plant toxins, and UV light, and enumerated the advantages of their use in such studies. Guppies were exposed by Scherf[116] in the dark to concentrations of 90 and 100 mg diethylnitrosamine/l for up to 7 months and found lesions, but no tumors, in organs of the peritoneal cavity. Scarpelli[117] studied the extent to which aflatoxin hepatocarcinogenesis in rainbow trout was modified by chronic inhibition of drug metabolism by *p*-diethyl aminoethyl diphenyl

...ropylacetate hydrochloride (SKF 525A). Intraperitoneal injections of SKF 525A prior to aflatoxin B_1 inhibited liver o-dealkylation and accumulation of smooth surface endoplasmic reticulum. Only one of 17 trout receiving SKF 525A prior to aflatoxin B_1 developed liver tumor. Sixteen of 30 fish not receiving SKF 525A developed hepatomas. Klingerman and Bloom[55] demonstrated sister chromatid exchange and sister chromatid differential by fluorescent and Giemsa procedure in the central mudminnow (*Umbra limi*) 5 to 8 days after injection with 800 μg 5-bromodeoxyuridine/g. The procedure was suggested for evaluating mutagenicity of water-borne chemicals.

Hematological effects of oral doses of PCBs in the European eel and rainbow trout were studied by Johansson-Sjobeck *et al.*[51] Eels given six repetitive doses of 1 000 μg/g showed a significant decrease in hematocrit after 2 weeks. Eels given a single 800 μg/g oral dose of PCBs had a significantly increased spleen somatic index and some cell enlargement in the liver. Additional data from these exposures were reported by Haux *et al.*[52] Increases in liver phosphate and liver triglyceride and a decrease in muscle water were the only significant effects found in the high dose repetitive experiments, while an increase in plasma chloride and a decrease in Ca were the only significant effects in the low single-dose experiment. Residues of PCBs 4 months after the low dose were 30 to 40 μg/g in treated fish, compared to 3 to 4 μg/g in controls. Westman *et al.*[53] studied the effect of a single oral dose of 1 000 μg/g of PCBs on the activity of delta-amino-levulinic acid dehydratase (ALA-D) and several other hematological parameters of rainbow trout. Blood ALA-D was significantly decreased 3 days after treatment, but returned to control levels during the remainder of the 21-day observation period. Spleen ALA-D activity was high during the entire observation period. No changes in hemoglobin or hematocrit were found. Lidman and Forlin[54] discussed the induction by PCBs of a "drug metabolizing system" in the liver of fish and its effect on the elimination of foreign compounds. Lidman *et al.*[56] investigated the metabolic effects on rainbow trout of a single sublethal oral dose of 800 μg PCB/g body weight. A liver residue of 15 μg PCB/g on a wet weight basis after 10 days and initial transient decreases (5 days post-feeding) in plasma K and Mg, with a late plasma increase in PO_4, 17 days post-feeding were found. A trend toward liver enlargement was also noticed. A 4-fold increase in o-demethylating activity of rainbow trout liver was found by Lidman *et al.*[56] 5 days after oral administration of 0.5 g PCBs/kg. Seven days after treatment, both cytochrome P450 and o-demethylating activity were increased, suggesting two or more steps in the induction of the drug metabolizing system of the trout by PCBs. Melancon and Lech[57] examined the bile of rainbow t[illegible] for polar metabolites after 24- and 48-[illegible] exposures to 5.0 and 5.4 mg C[illegible]-tetrachlorobiphenyl/l. A 4-hydroxy-TCB metab[illegible] was isolated and tentatively identified.

Pedersen *et al.*[58] compared liver homogenates from six strains of rainbow trout for hydroxylation of aniline and benzo(a)pyrene, reduction of p-nitrobenzoic acid to p-aminobenzoic, and the effect of 3-methylcholanthrene on stimulation of nitro reductase and aryl hydrocarbon hydroxylase. The study indicated some significant strain differences, suggesting caution in making comparisons of drug metabolism data of trout from different laboratories using undesignated strains. Possible induction by previous environmental or rearing conditions was suggested by the results of the stimulation experiments. The effect of a single intraperitoneal injection of 1.33 ml CCl_4/kg of fish on the activity of selected plasma and liver enzymes of rainbow trout was studied by Baccott *et al.*[59] The activity of all plasma enzymes, LDH, GOT, GPT, GDH, and GR, increased. The activity of liver LDH and GDH increased, while liver GOT and GPT decreased. The activity of plasma GPT and GR were considered the most sensitive indicators of CCl_4 damage. Bhattacharya and Mukherjee[60] investigated hepatic pancreatic protease and esterase activity in fish exposed to industrial pollutants. Effluents containing phenol, ammonia, sulfide, and Cu inhibited activity of both enzymes. The results indicated that low doses, where no mortality occurred, caused significant damage to the organs and inhibition of the enzyme systems. The metabolism of 2,5-diphenyloxazole (PPO) in trout livers was characterized by Ahokas,[61] and optimum conditions for metabolism of PPO by trout liver microsomes were established. Kobayashi *et al.*[62] studied the sulfate conjugation of phenol and pentachlorophenol (PCP) in liver slices or homogenates from goldfish, rainbow trout, spiny lobster, and shortneck clams, and compared the results with studies of rat liver. The sulfate conjugation by liver slices of the test animals was about one-thirtieth of the activity

or rat livers. Concentrations of PCP higher than 1.4×10^{-5} M were found to inhibit phenol conjugation. Ohmori *et al.*[101] reported that eel liver homogenates metabolize p-nitrotoluene more slowly than rat liver homogenates, possibly explaining the accumulation of toluene in fish.

Peake *et al.*[102] studied the effect of 0.15 percent and 0.30 percent ethyl alcohol in water on the elicitation and habituation of aggression in cichlids. Alcohol at a concentration of 0.15 percent made male fish hyperaggressive, and a concentration of 0.30 percent made male fish hypoaggressive, when compared to untreated controls. Goldfish were exposed by Fetsche *et al.*[103] to 7 to 150 mg cuthavine/l for 1 to 14 days. Significant increased general activity was found in shuttle box behavior tests. Stainability of the brain with the Timm histochemical procedure for heavy metals was also reduced with treatment, although atomic absorption analysis showed no significant changes in Zn and Cu concentrations. Hara[104] studied the olfactory responses of rainbow trout to amino acids under variable pH conditions, using electrophysiological methods. The effect of pH on the response was specific for each amino acid, but the maximal response for the most stimulatory acids occurred near their isoelectric points between pH 5 and 6. Cooper and Hasler[105] using electroencephalographic procedures, demonstrated a significant response by morpholine-imprinted coho salmon and rainbow trout to a stream sample containing 1.0 μg morpholine/l compared to unimprinted controls. Further, in field studies of coho salmon artificially morpholine-imprinted and released in Lake Michigan, Cooper *et al.*[106] confirmed the existence of odor imprinting and long-term olfactory memory.

Pulp and Paper. Fish stomach contents were studied by Pagel[107] in relation to the 1971 closing of a pulp and paper mill on Ticonderoga Bay, and the opening of a new mill in southern Lake Champlain, New York-Vermont. Ahokas *et al.*[108] investigated the mixed function monooxygenase system in pike (*Esox lucius*) as influenced by mixed pulp-chemical, factory-domestic effluent. This liver enzyme, known in mammals to respond readily to the presence of foreign chemicals, was found depressed in pike from a lake receiving the effluent.

Four static bioassays of neutral sulfite waste liquor were conducted by Griffin and West[109] using rainbow trout (*Salmo gairdneri*) as test animals. No differences were found in sensitivity because of fish size. The mean 96-hour TL_m was 0.62 percent. Lignin, sulfur dioxide, and ammonia were implicated as toxic components. Juvenile coho salmon (*Oncorhynchus kisutch*) blood clotting times and white blood cell plus thrombocyte counts (WBC + T) were determined by Iwama *et al.*[110] in response to dehydrobietic acid exposure. Exposures were made while the fish were exercised at low swimming speeds. Clotting times were increased after 48 hours of exposure, and WBC + Ts were reduced after 24 and 48 hours. These changes were not found after shorter exposures, and only at 0.5 body lengths/s swimming rate in the case of clotting time, and 0.5 body lengths/s and 1.05 body lengths/s for WBC + T. Swimming rates of 1.97 body lengths/s provided a level of resistance for some unknown reason. McLeay[111] studied the effects of pressure on changes in red blood cell (RBC) and WBC + T counts in juvenile coho salmon. Depression of the WBC + T count was found to be extremely sensitive to general acute stress, with statistically significant depressions occurring within 24 hours at 0.5 of the 96-hour TL_m for Zn, and 0.35 of the 96-hour TL_m for bleached kraft mill effluent (BKME) before treatment, and 0.51 of the 96-hour TL_m after treatment. Water extracts of western red cedar heartwood, bark, and foliage were studied by Peters *et al.*[112] for individual and combined toxicity to various life stages of coho salmon. The relative sensitivity of four other species, prickly sculpins (*Cottus asper*), steelhead trout, mayflies, and caddis flies was also studied. Heartwood lignans and bark extractives were found moderately toxic to coho salmon, but foliage terpenes and heartwood tropolones were more toxic, ranging from a 96-hour TL_m of 0.33 mg/l for coho salmon fry to greater than 9.8 mg/l for eyed eggs. Steelhead trout and sculpins were similar to coho salmon in tolerance to terpenes.

Rogers and Mahnad[113] subjected sockeye salmon (*Oncorhynchus nerka*) smolts to freezing of periodic samples of whole kraft mill effluents before and after model biobasin treatment. Resin acid content was reduced after treatment. Toxicity was associated with resin acids and unsaponifiable fractions and was influenced by the species of tree being pulped, with lodgepole pine producing more toxic extracts than Douglas fir and white spruce. The predominant toxicants in mechanical pulp mill effluents using an approximate spruce (75 to 85 percent), pine (15 to 25 percent), and fir (5 percent) mix were found by Leach and Thakore[114] to be resin acids, which ac-

counted for 60 to 90 percent of the overall toxicity of the effluent to rainbow trout. The bulgepole pine content of the mix was found important to the toxicity of the total effluent, contributing 40 percent of the total resin acids. Toxic units calculated from chemical analysis of the effluent were in good agreement with toxicities determined in rainbow trout bioassays.

Brown and McLeay[60] exposed starved juvenile coho salmon to neutralized treated and untreated BKME and found increased survival times after treatment by foam-stripping.

Relative growth rates of fishes inhabiting polluted and unpolluted sections of the Pearl River (Louisiana and Mississippi) were studied by Bronnick[61] before and after initiation of secondary wastewater treatment of a kraft pulp and paper mill. Growth rates were lower among blacktail shiners (*Notropis venustus*) and silvery minnows (*Hybognathus nuchalis*) from polluted water than from unpolluted water. Longear sunfish (*Lepomis megalotis*) and largemouth bass (*Micropterus salmoides*) from the two areas were not so affected. Sixteen months after secondary treatment of the effluent, blacktail shiners and silvery minnows no longer showed growth differences between the two areas. Jaak *et al.*[62] used a pilot-scale two-stage activated sludge system run in parallel with a conventionally-loaded single-stage system and found the toxicity of kraft bleachery effluent to be reduced more by the two-stage system than by the single-stage system. Effluents of hardwood processing were less toxic to juvenile rainbow trout than those from softwood processing.

McLeay,[63] proposed a short-term, low-toxicant volume bioassay procedure using residual air in sealed fish containers to indicate the onset of adverse toxicant concentration effects. The 24-hour and 96-hour TL_m values were also established using juvenile coho salmon and rainbow trout as test animals. Dehydroabietic acid, Zn, phenol, lindane, and neutralized BKME were tested toxicants.

DOMESTIC POLLUTANTS

Eutrophication of surface waters of Lake Tahoe's Indian Creek Reservoir was studied to evaluate the relationship between water quality and the reclaimed water exported by the South Tahoe Public Utility District (Lake Tahoe Area Council[64]). On the average, the reclaimed water contained from 15 to 24 mg ammonia/l, causing toxicity to rainbow trout (*Salmo gairdneri*) fingerlings. Other components of the system, such as N and P, and effects on plant life were discussed. Howell and Jones[65] studied the extent of pollution caused by three wastewater treatment plants on two streams in Jessamine County, Kentucky. Hickman Creek showed evidence of pollution for a greater distance than Jessamine based on the diversity of clean water indicator organisms, particularly darters (*Etheostoma* sp.) and stoneflies. Brynildson and Mason[66] described the extent and severity of kills of wild brown trout (*Salmo trutta*) in Big Roche A Cri Creek, Wis., caused by organic pollution from a primary waste treatment plant and documented the recovery of the trout population after construction of a new secondary treatment plant. After construction, production of brown trout increased above that in the unpolluted area of the stream. Effluent from the treatment plant apparently increased the fertility of the stream.

A simple model was developed by Wallis[67] to predict the impact of waste on a stable fish population. A model of this type, calibrated by field and laboratory determinations of the effect of a waste, could give order of magnitude estimates of changes in fish populations caused by waste discharges. Hendricks and Bluestein[68] projected the water quality and biological response of the South Platte River system, using water quality model Pioneer I and a biological "criteria vector" approach. With limited further development, the mountain streams would retain an essentially pristine condition and coldwater fishery and meet effluent limitations. Implementation of 1983 and 1985 limitations imposed by the EPA would produce water quality suitable for a warmwater fishery in the plains streams; however, severe habitat conditions in the plains would still be limiting. Davis[69] described an approach for setting effluent standards for the Glamorgan River, Wales, with a basic strategy of "maximization of the capacity of the river to assimilate waste but preserving appropriate multiple use function." Considerations for setting standards included water use classification, dilution, volume of the stream, physical character of the stream at the point of discharge, reaeration and self-purification capacity of the stream, metals, and other substances toxic to the fish in the discharge.

Gill and Tiwari[70] conducted static bioassays with *Puntius ticto* in wastewater effluents from the city of Ludhiana discharged into the Budha Nullah, India. The 24- and 48-hour TL_m values were 33 and 31 percent effluent. Bioassays with fingerling sockeye salmon (*Oncorhynchus nerka*) were conducted on primary

effluents from three wastewater treatment plants by Mertens and Servizi.[113] The 96 hour TL_m for the plants ranged from 17 percent to 35 percent effluent. Multiple regression analysis of mean geometric survival times for data from one plant indicated anionic surfactants and un-ionized ammonia positively correlated, and alkalinity and Fe negatively correlated with acute toxicity. However, not all toxicity was accounted for by the substances measured. Fava and Tsai[115] and Tsai and Fava[116] investigated the immediate avoidance reaction of blacknose dace (*Rhinichthys atratulus*) to chlorinated and unchlorinated wastewater, and to free chlorine, chloramines and ammonia in laboratory tests. Threshold avoidance concentrations to free chlorine, chloramines, and chlorinated wastewater were 0.01, 0.18, and 0.13 mg/l as total chlorine. No avoidance reactions were caused by ammonia and unchlorinated wastewater. Based on the similarity of concentrations causing a reaction and the similarity of the response, chloramines were suggested as the constituent of chlorinated domestic wastewater causing avoidance reactions of fish observed in earlier studies.

RADIOACTIVE POLLUTANTS

Rauchaly *et al.*[101] studied the effects of gamma irradiation of 100 Roentgens (R), 200 R, and 400 R on the Nile catfish (*Clarias lazera*), and at 500 R and 1,000 R on *Tilapia nilotica*. Significant concentration-related reductions in erythrocyte counts and hemoglobin content occurred in catfish following irradiation. Recovery of these parameters took place in about 4 weeks at 200 R or less, but not until about 10 weeks at 400 R. Creatinin levels were little or not affected. Growth rates and protein changes were also reported after irradiation. Irradiation of *Tilapia nilotica* at 500 R and 1,000 R resulted in an initial hyperglycemia response, following a continual decline until an hypoglycemia was indicated after 24 hours. Glycogen stores in the liver followed a similar trend at 1,000 R, but not at 500 R. Muscle stores were apparently unaffected at 500 R, but were depressed after the 1,000 R exposure. Free reduced sugars declined after irradiation, as did hydrolytic enzyme activities.

Till[102] exposed embryos of carp (*Cyprinus carpio*) and fathead minnows (*Pimephales promelas*) to isotopes of Pu and U to determine the chemical and radiological toxicity and distribution within the embryo. The effects on carp of both elements were less than on minnows. Hatching failed completely when embryos were exposed to 20 mg ^{239}Pu/l, but hatching was not affected at 80 mg ^{239}U/l and ^{239}U/l. ^{239}Pu toxicity was probably chemical rather than radiological. Egami and Hama[103] studied the effects of radiation on hatchability of medaka (*Oryzias latipes*) embryos when exposed at different embryonic stages, at different doses, and at the same dose but at different dose rates. The effects of exposures also were observed on the viability of gametes within the parent fish at different times before fertilization. Early embryo stages were consistently sensitive to radiation with later stages less so, but intermediate stages varied in sensitivity. High dose rates (33.3 R/min and greater) were more deleterious than low rates (1.7 R/min and less). High temperature intensified adverse effects at low dose rates. *In vivo* irradiation of gametes inhibited hatchability. Spermatozoa were more sensitive than ova, and recovery with time post-irradiation was more complete within 5 days in ova than among spermatozoa. Newly-hatched medaka were exposed to x-rays (1,000 R) by Hamaguchi and Egami.[104] Exposures were made 3, 10, and 15 days post-hatching, followed by serial sectioning at 1 to 30 days after the exposures. Female germ cell counts were then made to determine effects. Germ cells in the diplotene stage were killed within 24 hours after irradiation. Germ cells in the leptotene, zygotene, and pachytene stages failed to enter the diplotene stage before degeneration. Oogonia were killed immediately or underwent normal development up to the pachytene or diplotene stage and then degenerated. Hyodo-Taguchi and Egami[105] exposed cold-acclimated medaka held at 0° to 5°C for 100 to 1,000 rads of x-rays. At this temperature, the gonads were found to contain primary spermatogonia Ia, Ib, and secondary spermatogonia II. Irradiation had little effect on Ia cells, suggesting little mitotic activity. Marked effects on Ib cells were seen with significant cell death resulting. Post-irradiation observations, after a temperature increase to 25°C, showed a recovery in the number of Ib cells believed to be derived from surviving Ia stem cells. Person and Raymore[106] studied post low-level ionizing radiation effects on Atlantic salmon (*Salmo salar*). Effects reported included changes in body weight, numbers of sexual cells, and reversal of sexual trend in males. The trend in effect was related to the dose and the state of the reproductive system at the time of exposure. Juvenile rainbow trout (*Salmo gairdneri*) were exposed by Strand[107]

for 80 days as embryos to 0, 0.04, 0.4, 4.0, and 40.0 rads of tritium. Reduced immune response was found in fish exposed to the two highest doses. Hatchability, incidence of abnormalities, latent mortality, and depressed growth were not observed at any of the tested tritium concentrations, which were 5 to 8 orders of magnitude above present tritium concentrations in aquatic environments and 3 to 4 orders of magnitude above estimated future environmental levels. Pitkyanen and Zaitsev [107] discussed the means by which a dose of ionizing radiation, specifically ^{90}Sr, ^{90}Y, and ^{137}Cs, can be accrued by fish eggs, depending on the degree of previous exposure of the parent fish, incubation time, distribution of the eggs in the water body (whether pelagic, benthic, or suspended), and the radiation-absorbing nature of the substrate upon which the eggs were supported. Eggs on sandy bottoms may receive only 0.01 times as much exposure as the same eggs on highly organic bottoms, and eggs that incubate off the bottom by nature of their specific gravity, adhesiveness, or deposition on supporting objects may receive 2 to 75 times less exposure than those resting on the bottom.

Harriman *et al.*[108] found that fish were the primary potential route of radionuclides from the Gundremmingen Nuclear Power Plant in Bavarian Germany to man. Average consumption of fish flesh in the Federal Republic of Germany ranges from 10 g person^{-1} d^{-1} to 180 g person^{-1} d^{-1} providing potential human doses of 0.18 to 97 mrem y^{-1}. Blaylock and Witherspoon [109, 110] investigated the release of radioactive wastes to aquatic environments associated with seven processes of the nuclear fuel cycle: uranium mining, uranium milling, conversion facilities, uranium enrichment, fuel fabrication, light-water reactors, and fuel reprocessing, to assess the impact of estimated dose on aquatic biota. Mining and milling processes were found to be by far the greatest contributors to environmental radiation contamination. Even with long-term irradiation data, now largely unavailable, radiation effects at the population level probably would not be detected even at mining and milling sites.

Eyman and Kitchings [111] exposed bluegill (*Lepomis macrochirus*) and channel catfish (*Ictalurus punctatus*) held at 13° and 25°C to ^{137}Cs-contaminated sediment materials, kaolinite, montmorillonite and illite. Exposures were by gastric injection. Sediment type was found to have an important effect on assimilation and elimination. Illite, the most organic sediment, released less ^{137}Cs to the fish. At 13°C, assimilation was generally greater than at 25°C, but the temperature relationship was only significant with montmorillonite. The importance of sediment type in relation to nuclear power plant siting was stressed in their conclusions. The distribution of radiocesium in water, sediments, and biota was studied and modeled by Jinks.[112] Mean concentration factors were reported for marine fish of about 80 and for freshwater fish of over 1 000. The primary source of uptake was from that dissolved in the water. Sediments were found to be a secondary source, being most important in benthic feeding fishes. Uptake was also found inversely related to ambient water K content, and directly related to the trophic feeding level of the fish. Freshwater piscivorous fish were found to contain about twice as much as prey fishes. Concentration and accumulation factors for ^{137}Cs in the edible portion of fishes were studied by Ettenhuber and Roehnsch.[113] Fish were taken from the commercial catch from the Greifswalder Bodden (a bay of the Baltic Sea), Stechlinsee (an oligotrophic lake), the Elbe and Elde Rivers (industrially polluted waters), and the Muggelsee (a river reservoir). Variations were found between species and within species from different water bodies. Buyanov and Antonenko [114] studied the uptake and concentration of ^{137}Cs by aquatic life at several trophic levels in three northern freshwater environments. Concentrations in fishes from higher trophic levels were higher than in organisms from the first consumer level, and concentration was inversely related to mineralization of the ambient water. The trophic level effects indirectly demonstrated the dominant role of food as the route of entry into fish. A model for predicting accumulation and retention of trace contaminants obtained through food ingestion in aquatic consumers was tested by Ehrmoot and Eyman.[115] Results of this model test indicated that at the feeding levels used, the model will predict ^{137}Cs accumulation in bluegills in acute exposures, but appears to overestimate the exposures of this contaminant beyond 10 days.

Wahlgren and Marshall [116] studied the distribution and movement of long-lived radionuclides of ^{137}Cs, ^{239,240}Pu, ^{241}Am, ^{134}Cs, and ^{85}Sr in Lake Michigan. Uptake of ^{85}Sr was found inversely related to calcium content in the water. ^{137}Cs in a given trophic level was directly related to the ^{137}Cs/K ration, and plutonium concentration was related to the soluble silica level. Concentration factors in edible fish from Lake Michigan were 4 to 40 for ^{239,240}Pu,

180 to 480 for ^{181}W, and 15 to 67 for ^{85}Sr. Plutonium concentrations in fish tended to decrease by about one order of magnitude in going from benthic feeding forms to plankton feeders to piscivores. The uptake and elimination of radiotungsten by black bullheads (*Ictalurus melas*) was examined by Rancitelli [11]. Bullheads accumulated radiotungsten from both food and water, with activity reaching a plateau after fish were in tagged water for 4 days. The bone, skin, flesh, blood, and gills contained most of the whole body activity after 1 day of uptake from tagged water. After 8 days, the flesh, gills, bone, and gut together contained 78.6 percent of the total activity. The bone had the longest biological half-life (8.0 days) of the tissues examined and contained 60.8 percent of the whole body activity after 16 days of elimination. Tissue residues of ^{90}U and ^{226}Ra were studied by Justyn and Lusk [12] in fishes inhabiting streams in northern Bohemia without U mining and milling and before and after mining and milling activities had commenced. By maximizing the possible legal catch and consumption of brown trout (*Salmo trutta*) by an individual fisherman harvesting a contaminated stream, it was possible to be exposed to about 4.58 percent of the stated safe U intake per year, and about 183 percent of the safe annual ^{226}Ra intake. Low level plutonium emissions from the Rocky Flats Plant of the U. S. Energy Research and Development Administration reached a small stream in which bullhead minnows (*Pimephales vigilax*) were found by Thompson [13] to concentrate the Pu in 0.90 to 7.5 times that in water. Nicolas and Kirchmann [14] studied the uptake, tissue residue distribution, and clearance rate of ^{54}Mn and ^{65}Cu in feeding, starving, and dead freshwater minnows (*Phoxinus laevis*). Fed fish accumulated more of both isotopes and did so at a faster rate than did starved fish, but dead fish accumulated 10 times as much as live fish. The higher concentrations in dead fish were suggested to be caused by absence of waste removal processes and uptake regulatory mechanisms.

Berg and Weiss [15] studied the transfer of ^{65}Zn from sediments to chironomid larvae to a small freshwater fish, *Haplochromis burtoni*, as influenced by the presence of Cd. Cadmium was in the form of the ^{109}Cd isotope and was present in the chironomid larvae fed to the fish and also in the aquarium water. Cadmium strongly inhibited ^{65}Zn exchange from the sediments to the larvae; however, at the concentrations employed (0, 10, and 50 µg/l), Cd did not inhibit ^{65}Zn exchange to the fish. Sukalskaya *et al* [16] presented a two-compartment model of ^{85}Sr exchange in water and freshwater fish eggs. The model described kinetics of ^{85}Sr accumulation in and elimination from the eggs; it also compared ^{85}Sr exchange with that of ^{45}Ca. The separate and combined effects of ^{85}Sr and the organophosphate pesticide bacudin were studied in the bighead (*Aristichthys nobilis*) by Alekseenko and Frolopenko [17]. The effects measured were percent mortality after 10 days, O_2 uptake rates, and ^{85}Sr tissue residues. Mortalities and respiration rates were higher among fish exposed to both pollutants together than in either alone. Respiration increased in the presence of both toxicants at about the same rate, but, in combination, the increase appeared additive. Tissue residues of ^{85}Sr in the fish were reduced 39 percent by the presence of the pesticide.

OTHER POLLUTANTS

Moyle [18] compared data on channelized and unchannelized sections of Rush Creek, Modoc County, Calif., and found fish biomass in the channelized section to be less than one-third that of the unchannelized portion. The average size and number of rainbow trout (*Salmo gairdneri*), average size of brown trout (*Salmo trutta*), and the numbers of the rare endemic modoc sucker (*Catostomus microps*) were less than the same species in the unchannelized section. King [19] studied short-reach channelization associated with highway bridge replacement on six central Iowa streams. Dissolved oxygen (DO) and turbidity were apparently unaffected, and there was only minor impact on fish and fish food organisms. Fish catches in recently channelized streams lacking cover were significantly less than in unchannelized sections, but catches were similar in older channelized streams. Stream morphology and gamefish populations of the St. Regis River, Mont., were studied by Lund [20] during the summers of 1973, 1974, and 1975 to determine the effects of stream channelization caused by highway and railroad construction. Results showed that mitigating structures (jetties and random rock clusters) were effective in providing fish habitat comparable to unaltered sections. Fish populations in new channels usually stabilized in approximately 1 year. Trout populations in "old" altered sections with no mitigation failed to recover to unaltered section levels. Kelly [21] studied three segments of the Luxapalila River in northeastern Mississippi and northwestern Ala-

basins to determine if differences in fish species diversity existed between channelized and unchannelized segments. The unchannelized segments of the river were the most diverse, and the recently channelized sections the least diverse. Bottom fishes were the most severely restricted forms in the recently channelized areas, while open-water forms were greatly reduced in the older channelized sections.

Hazels and Cobb [42] found that sand slowed the emergence and reduced the number of fry emerging when brook trout (*Salvelinus fontinalis*) alevins were buried in laboratory troughs in spawning gravel which contained 0 to 25 percent sand. The percentage of fry emerging decreased significantly with increased sand composition. After road construction, increased logging, and severe floods deposited excessive sediment from 1950 to 1960 in the South Fork Salmon River, Idaho, Platts and Megahan [43] evaluated trends in the size of riverbed surface materials. A large decrease in fine sediment and a large increase in gravels improved spawning of chinook salmon (*Oncorhynchus tshawytscha*) and steelhead trout (*Salmo gairdneri*) from 1966 to 1974. A moratorium on logging and road construction activities and a watershed rehabilitation program during this latter period demonstrated that sediment damage was reversible. Branson and Batch [44] reported that continued siltation from strip mining operations in two tributary streams of the North Fork of the Kentucky River prevented recovery of fish populations. All species were forced downstream and six species were absent. Populations of the creek chub (*Semotilus atromaculatus*) were on the increase, possibly reflecting the removal of competing species. Equations developed by Kuhl [45] described the effects of low concentrations of tin and increased levels of fine sediment on survival to emergence and fitness of chum salmon (*Oncorhynchus keta*) fry. Survival to emergence decreased 1.56 percent for each 1.0 percent increase in sand content. A selective mortality against fry of a larger size was also suggested in gravel containing high amounts of sand. Swenson and Matson [46] measured growth and survival of larval lake herring (*Coregonus artedii*) and found larvae were not influenced by red-clay turbidity at concentrations which included the normal turbidity range of the red-clay area in western Lake Superior. However, larvae in the higher suspensions were distributed closer to the surface of the test chambers. Green sunfish (*Lepomis cyanellus*) were exposed to bentonite clay suspensions at temperatures of 5°, 15°, 25°, and 35°C by Herkel and Pearson.[47] Oxygen consumption rates were not affected by turbid suspensions up to 1,500 Formazin Turbidity Units (FTU) at any of the four temperatures, while ventilation rates increased 50 to 70 percent at 1,013 FTU at 15°C and 808 FTU at 25°C. Increased ventilation rates under highly turbid conditions were a strategy for maintaining constant O_2 uptake.

Thomas [48] observed that migration of chinook salmon fry was stimulated by rising water temperatures and increased turbidity, but that doubling the water flow did not increase migration. June [49] sampled young-of-the-year fish stocks from 1960 to 1974 in the Lake Oahe storage reservoir of the Upper Missouri River. Yellow perch (*Perca flavescens*) was the most abundant species, with abundance increasing during reservoir filling and declining later. Emerald shiners (*Notropis atherinoides*) ranked second in abundance, and followed the same pattern. Reduction and degradation of littoral spawning and nursery habitats were probably responsible for the general decline of these species after filling of the reservoir. Gabelstein [50] found that fishes of all four riverine in the upper Shetucket River system in southern New England were affected to some degree by man. The fluctuating water levels from power generation at Scotland Impoundment probably had a greater effect on fish populations downstream than any of man's other actions. Hydroelectric power generation operations created high water velocities with daily flow reversals and water level and temperature fluctuations in Leesville Lake, Virginia, and Bennett [51] found that water velocity during power generation was the single most important factor affecting distribution of spawning nests of centrarchid fish in the main lake. Hatching success of naturally-spawned bluegill (*Lepomis macrochirus*) eggs exposed to air during night and daytime hours decreased with increased exposure. El-Sadly and Libosvarsky [52] found that a gradual decrease in fish production and a shift in favor of *Tilapia* species has occurred since 1966 in Lake Burullus at the mouth of the Nile River, Egypt. Erection of the High Dam at Aswan has controlled flood water, with a reduced input of fish from the Nile River. It was noted that overfishing of the lake may also have played an important role in this species shift and reduced production. Since the construction of two more dams on the Snake River in 1969 and 1970, average travel time for yearling chinook salmon migrating 370 km

downstream has increased by an additional 16 days in low flows and 11 days in moderate flows. Beutler and Raymond[55] discussed the problems of predation and disease during the impoundment and the possibility of holdover in the reservoirs so that some juveniles would perhaps never reach the sea.

Results during 10 years of habitat management in Big Roche-a-Cri Creek, Wis. were documented by White.[61] An increase in water volume resulted in an increase in the brook trout (*Salvelinus fontinalis*) population and narrowing the stream and providing overhead cover helped ameliorate unfavorable effects of low flow. Hunt[60] monitored responses of wild brook trout and found that maximum number and biomass and number of legal trout did not occur until 5 years after completion of instream habitat development. A start-pause-finish sequence of research to evaluate effects of environmental perturbations was suggested.

Mills[60] reported that better land management practices during lumber operations where stream courses are involved have resulted because of new legislation and public pressure, particularly in Oregon and Washington. Directional tree falling and skyline yarding make it possible to leave streams and banks intact. Forest roads continue to pose serious problems of sedimentation, however. Sutherland and Ogle[57] investigated the possibility of physical damage to the eggs of chinook (quinnat) salmon by the passage of jet boats through spawning areas in New Zealand. The passage of the boats gave rise to fluid pressures which were different from normal hydrostatic pressure, and the yolks were easily ruptured by the physical shock.

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